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THE
LARYNGOSCOPE.

VOL. LXII OCTOBER, 1952. No. 10

THE 1952 HERBERT STANLEY BIRKETT LECTURE; "A CRITIQUE OF PRESENT DAY METHODS OF HEARING-TESTING."*

D. E. S. WISHART, M.D.,
Toronto, Canada.

We are assembled to honor the memory of a name great in the medical annals of McGill University, Montreal and Canada. To those responsible, I express my sincere thanks for inviting me to deliver the 1952 Herbert Stanley Birkett Lecture.

Those of you who have read the admirable obituary, penned by your own Dr. W. J. McNally and published in the *Annals of Otology, Rhinology and Laryngology* in 1942, will be astonished at the multitude of Dr. Birkett's activities and accomplishments. He was a truly amazing man and his success, phenomenal. Unquestionably, he is one of whom you must always be proud.

I had the pleasure of being in his company many times, for he was a close friend of my father. They were at McGill at the same time, in medicine, and in the same fraternity. They were alike in their hatred of what their consciences knew to be wrong. Dr. Birkett jealously guarded and upheld the integrity of otolaryngology. To me, he was a shining example of what a courteous gentleman should be. I remember him most for the way he seemed to consider charitably that some of my faults were prompted by better motives.

*Read at McGill University, Montreal, Canada, April 25, 1952.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, June 15, 1952.

He was a very great Canadian doctor and I deeply appreciate the honor of delivering the lecture by which his name is perpetuated.

* * * *

Ever since the teaching of medicine commenced in Montreal the name of McGill University has been world famous. Some of the hospitals in this city are now about to tear down the buildings which have served them so well and are engaged in planning that the new structures shall be worthy of the great traditions of the past.

One of the problems which you as otolaryngologists must settle for good or ill is how you shall plan for proper attention to hard-of-hearing adults and children.

Hearing Testing Unsettled.

The testing of hearing is in a muddled state.

The formerly accepted tests of hearing have been proved so inaccurate that they have been abandoned almost everywhere. In their place came the pure-tone audiometer for a time, but it has now become apparent that it is impossible from the study of an audiogram to determine how well a patient hears speech. The result has been the re-establishment of measured speech-hearing-testing.

In the enthusiasm for this, certain great hospitals have set up establishments for testing hearing which are costly and complicated. The claim is made that the many elaborate tests thus made possible provide information of value. There is, however, another side to the picture.

In certain of these hospitals only a very few technicians — in some only one — can or are allowed to run the audiometers and use the soundproof room; the examinations are lengthy and may take five hours or more to complete, and they are fatiguing to the patient. The reports issued are couched in such terms that few if any of the attending staff can understand them; it is rarely claimed that they can be used for comparison with results obtained on other occasions; and to

crown all — some of the government boards to whom these elaborate reports are made for purposes of determining pensions, etc., completely disregard these audiological findings and base their decisions on unmonitored conversational voice.

In what follows I hope to convince you that certain prevalent fashions in the testing of hearing are wrong and to indicate certain other things which are essental.

Purposes of Hearing Tests.

Hearing tests are performed for nonmedical as well as for medical reasons. Examples of the former are — the determination of the suitability of employees for certain jobs, the effect of workshop noise on the employees, the screening out of the school children who should be referred to a doctor, etc. These can be performed by technicians or by the specially trained technician — the audiologist.

On the other hand, hearing tests for diagnosis, prognosis and evaluation of treatment should be performed by the otologist. For he serves patients who wish to know why their hearing defect exists, what the future holds for them and what can be done to alleviate the impairment. The main essential of a hearing test is measurement.

Otologist Squeezed Out by Audiologist.

Even as short a time as 15 years ago there were few ear, nose and throat clinics where there was any provision whatever for the testing of hearing; the few hospitals where an attempt was made to provide proper facilities were distinctive because of such provision. Now, however, great changes have occurred, and there are many cities where one or more hospitals provide suites of rooms exclusively set apart for the testing of hearing, with elaborate nearly soundproof rooms, great installations of expensive audiometric equipment and a staff of trained technicians headed by an audiologist. These elaborate schemes have arisen from the belief that the testing of hearing is passing from the hands of the practicing otologist and will be confined strictly to expensive hospital departments devoted to the testing of hearing.

In many of these a considerable hospital distance separates the hearing suite from the otologic clinic. In some there are peculiarities such as the following: visits by any otologist may be infrequent; the hearing establishment may be run exclusively by the audiologist, his special assistants, his technicians and his nurses; in the ear, nose and throat clinic there may be no provision whatever for the testing of hearing by the clinical otologist, and the latter may depend entirely upon the reports sent from the audiological department.

The testing done in some of these departments is most elaborate and time-consuming — so much so, that in very few is there one attending otologist who is capable of performing the tests and in the majority there is no otologist who could afford the time involved.

Audiological Work-Up Tests Without Validity in a Clinic.

Many new hearing tests have been described and some of them strongly supported. Every audiological clinic has a formidable list of tests.

Guild[2] says: "At least 10 to 12 hours of time per patient would be needed to administer all the hearing tests now regarded by one or another investigator as of interest and value. The apparatus to make possible the administration of these tests would cost a minimum of several thousands of dollars. This figure does not include the cost of the special rooms needed for satisfactory use of the testing apparatus, nor does it include the salaries of specially trained technicians to use the apparatus, or payments to skilled electronics engineers to keep the various pieces of apparatus in proper operating condition."

The supposition that these tests are of clinical value is not correct.

In a lawsuit a physician stated he had done all the tests. This drew the sage retort that a medical man who would do all the tests would be either naive or unscrupulous. A wise and skilled medical man should perform only those tests which he knows will add to the knowledge of the patient's troubles, which are reliable and which are valid.

Knowledge of any part of the body is based upon a correlation of function with the gross and microscopic appearance. Real knowledge regarding hearing defects depends upon evidence from innumerable cases for which there has been adequate history, painstaking examination, accurate hearing tests, plus descriptions of the gross and microscopic pathology.

The amount of such material available for critical study is not great and few teaching centers possess any whatever. The evidence from a very good source of information is that "histopathological evidence does not exist to support any of the hypothetic interpretations that have been proposed as explanations between functional differences observed in the fitting of hearing aids on persons with otherwise similar degrees and types of deafness."[2]

Guild[2] has written three condemning paragraphs which have not been controverted:

"The information obtained from the elaborate tests or batteries of tests is primarily of research interest. Until the significance of the data obtainable by these tests has been evaluated in terms of lesions that otherwise would be overlooked or in terms of therapy that otherwise would not seem indicated, clinical otologists should not be urged to use them or made to feel they are out of date if they do not.

"In other words, it is time for a clear distinction to be made between tests for research purposes and those for clinical purposes. Nothing is gained clinically by the making of hearing tests that cannot be interpreted with reasonable certainty in terms of causative lesions.

"All too often, a diagnosis, so-called, amounts to nothing but a statement of the kinds of hearing defects found by the tests made. Such a statement, no matter how elaborate the battery of tests used or how detailed the report of the data obtained, is merely a description, and not an interpretation of hearing tests and has no medical usefulness whatever."

The establishments which I have criticized have a place in any institution devoted to research into problems of hearing

and of psychology, but are unnecessary in a doctor's office or in a hospital clinic. In these, there should be facilities which provide data that may be used for the good of the patient and the information of the otologist. A hospital clinic may have a research unit attached to it, but, as this unit cannot provide the information which the clinic and the otologist require, it is not necessary for hospitals to provide it, unless the hospital can afford the extravagance of personnel for a research program.

Interference with Clinic Is Wrong.

A great research unit and its personnel must not be allowed to interfere with the necessities of the hospital clinic.

Essentials for a Clinic.

The essentials of a satisfactory method of mechanically measuring the hearing are — accuracy, quickness, and a technique which can be exactly repeated at a later date. The apparatus should be as simple as possible, rugged, not likely to get out of order, and constant in performance.

Restoration of Otologist to His Proper Function.

I believe that the hearing facilities should be in the ear, nose and throat suite and that they should be such that any of the attending staff — providing he trains himself to operate the machines — can, when he desires, test the patient in whom he is interested. I do not believe that the day of the otologist is over.

Plan at the Hospital for Sick Children.

We have tried to provide such conditions at our new hospital in Toronto in the following way.

Our out-patient clinic has been so designed that the entire suite of rooms is at the service of the Hard-of-Hearing Clinic every afternoon. Certain of the rooms are available to the Hard-of-Hearing Clinic every morning.

The suite consists of five examining and treatment rooms, a hearing clinic room with an adjoining observation gallery, a

hearing test suite consisting of an anteroom and two nearly soundproof rooms, a research room, a secretary's office, an office for the chief of service and a small waiting room.

Our work roughly consists of the following: interviewing the parent, the taking of an adequate history, the physical and special examination of the patient, the estimation of the patient's hearing, and if treatment is possible, the provision of this treatment. Whether treatment is feasible or not, the parent is given detailed advice regarding the home care of the child, and the patient is referred to the best available educational instruction.

The work is carried on by two members of the staff, three Fellows, a secretary and the chief of service. We expect to obtain an experienced teacher of the deaf.

The clinic has been placed next door to the social service department and across the hall from the department of psychological medicine and the speech clinic, with all of whom we work closely in the follow-up of our patients.

Our hardest problem is the testing of the hearing of children 12 months to three years of age. In the hearing clinic room, little children who are extremely hard-of-hearing are examined with special testing devices. These are the Peep Show, the Melody Master and various game-methods.

Our Peep Show was evolved from the thumbnail sketch which accompanied an article by Dix and Hallpike[3] in the *British Medical Journal*, Nov. 18, 1947. From that we built an apparatus which would produce a sound when a child was looking at a picture through a peephole and provided a screen, behind which the apparatus and the operator of the machines were hidden. This original model required two or more workers to handle the machine and child.

During the evolution of our final model I visited Dr. Norton Canfield, of New Haven, and received his generous help. I did not copy his machine, but I did adopt his excellent idea of centralizing all the mechanism on one control board. Only one operator is required to handle our machine and the patient.

In this Peep Show we can use either a colored movie or colored still pictures. We find that the movies captivate most of our little patients, but occasionally a very timid child has to be tested with our set of still pictures.

Our second device for interesting children in sound is a Melody Master, which consists of a gramophone, headphones and microphones. The children gradually are persuaded to wear earphones through which they hear any gramophone record which suits our purpose. The mother assists by using a duplicate set of headphones and by endeavoring to speak to the child through a microphone. The method is of only slight value at the initial visit but is very useful after the child has been attending for a number of weeks. It is a pleasurable way of teaching the child to use earphones, for until this has been achieved the child cannot be tested with our speech audiometer.

A much smaller portable microphone and headset is available for demonstration to parents who desire such apparatus for use at home in the instruction of their child.

We have also many animal toys and peg-toys which can be be used to demonstrate to the mothers game-methods of conditioning their little deaf children to various sounds. All of these games are similar in their basic purpose, which is that by means of a slight reward the child learns that a funny stimulus it receives (namely, sound) has a meaning.

These four schemes arouse no antagonism or fear. The time consumed in testing is sometimes great, but in the end it is rewarding.

Special cupboards contain toys for the amusement of the children and the various forms of printed advice for the guidance of parents. Gradually we hope to acquire such a number of pamphlets and forms that we can meet the problems of any parent.

We advise parents of very young children to take the correspondence course of the Tracy Clinic.

Hearing Test Suite.

Two heavy doors make the hearing test suite soundproof except for very extreme noises. Ordinary noises in the adjoining rooms and halls and outside do not in any way interfere with the patient or examiner.

Two windows of unique construction look from the anteroom into the hearing-test rooms. Through these an observer in the anteroom can see all that is going on in the hearing-test room without those inside being aware that they are being observed.

This has great value in allowing an observer to learn how various hearing tests are performed, and a parent to see exactly how its child is being handled, without the child being distracted by the person in the anteroom.

The first hearing-test room has in it a double set of instruments. One set, consisting of a high-fidelity gramophone audiometer and a pure-tone audiometer, is for the use of the clinicians. The second pure-tone audiometer and a much less expensive gramophone audiometer are for the use of the young doctors who are learning how to do hearing tests. Masking is always used.

The second hearing-test room is smaller than the first and contains two instruments: a pure-tone audiometer and a special gramophone audiometer. These are for the use of the chief of service and his assistant.

We use three types of audiometers. The pure-tone audiometers are the last designed by the Western Electric Company. They are the best of their type and are known for their simplicity, reliability and durability. The special gramophone audiometers are instruments of unusual fidelity and were specially designed in Prof. Solandt's department in the School of Hygiene, Toronto. They are simple and accurate and full directions are provided so that any member of the clinic can operate them. These machines probably will be able to play any type of gramophone record which may be devised. The third machine is the Baldwin Gramophone Audiometer, — a recently marketed elaboration of the old 4-A Audiometer.

In our research room, we have a duplicate of the Johns Hopkins' skin resistance machine for the testing of the hearing of young infants. One of our Fellows is engaged in a three year investigation of the Baltimore claims.

All of these physical facilities are close to rooms where every type of clinical examination and treatment is available. The otologist who is to examine and treat the child and advise the patient is expected to attend every step. For the child must learn to trust him implicitly.

Statements Regarding Techniques.

Now I must direct your attention to certain details regarding technique which we find of great importance in the testing of children's hearing.

Re Pure-Tone Testing.

To obtain results when testing the hearing of a very young child, the child's apprehension must be removed, his interest aroused and his cooperation obtained and held.

This is difficult to achieve when using the pure-tone audiometer, — for the child has little interest in pure-tones.

The commencement of pure-tone testing is done with the Peep Show. It is an invaluable first approach. The child can operate it without instruction. The picturesque little house at once disarms the child's fears and arouses his curiosity. He naturally looks into the doorway — but he sees nothing: for the black hallway is in darkness; however, what is a doorbell button for but to be pressed? The result is a pleasant discovery. A picture appears which is moving. The moving pictures of farm activities are pleasing and have dramatic attention-holding appeal. The desire to see what comes next keeps the child's cooperation. When his little arm is too weak to press the doorbell button the patient can rest his arm on the ledge and press the doorstep.

Over the doorway is a small red fanlight and behind the gable decoration is a loud-speaker. When the child presses the button a flashing light appears in the former and from the latter comes a musical tone synchronous with the flashing light. The light and the sound can be altered at will by the operator.

Four discrete frequencies—500, 1,000, 2,000 and 4,000 c.p.s. —are provided. The rate can be varied and also the relative lengths of the off and on periods.

In this test the child learns that when he responds to the flashing light by pressing the button he is rewarded by a continuation of the pleasing moving picture and also that when the light is not flashing, pressing the button produces no picture.

While this lesson is being learned, the operator is experimenting with different intensities of the four available sounds. The control board can be operated without the child's being aware of it. Sometimes the child will suddenly investigate the gable, thus betraying the fact that he has located the source of the sound. Sooner or later the operator switches off the light and in a favorable case the child continues to press the button whenever the sound is being produced.

The child has discovered that a certain peculiar sensation has a meaning; that, if, when he perceives that sensation, he presses the button, he will get the reward of more pictures. The pleasure the child gets is evident to all the observers and frequently brings tears to the eyes of the mother, who up to that point has believed her child could not hear. The child has discovered that sound has a meaning and hope has been kindled in the mother's heart.

The doctor, on the other hand, has formed a rough idea of the degree and extent of the hearing loss by having observed the intensity of each note that has reached the child's intelligence. A very rough audiogram for four tones can be obtained in some cases.

This happy result can be the early stage of pure-tone testing for the little patient. If we had proceeded differently and had introduced the child to the pure-tone audiometer we would probably have aroused resentment and fear, for a child is not attracted by the uninteresting sounds of a pure-tone audiometer.

To keep the child's attention it is necessary that there be as few distractions as possible. As a rule, no one else is in the room except the child and the otologist; however, it is often necessary that the mother sit beside the child. Observers are accommodated in the observation gallery where they can hear and yet are not seen.

Testing Older Children with Pure-Tone Audiometer.

We do not attempt testing a child with the pure-tone audiometer until much later in its clinic life. We do not use it until the child's fears have been eliminated, until it trusts and obeys its instructor.

In using the pure-tone audiometer we have given up having the patient press a button. The action is distracting and takes time. We have the child raise his finger when he hears the tone and drop it when the sound ceases. The child sits with his back to the examiner and the latter watches the action of the finger and from it judges his patient's interest, attention, cooperation and alertness.

We bother not at all with speech tests performed with selected test lists designed to show up losses of hearing in different parts of the frequency range. The best way to test frequency hearing is to use frequencies as stimuli. One cannot infer what speech hearing is from an audiogram.

We make all tests very short. The time cannot be stated exactly. A young child ought not to be in the soundproof room longer than a few minutes at a time. Loss of interest on the part of the child will be shown in the gradual drooping of the signal finger, the delayed response of the finger, the swinging of a foot, etc. As soon as the child's interest or cooperation flags, we stop the test and leave the room.

Re Speech Testing.

Our testing of speech is simple. We are not convinced that any of the newer and more elaborate methods of using speech in the testing of hearing are applicable to as wide a variety of patients as is the simple phonograph test.

We use the high-fidelity gramophone and the Western Electric recording K.S. 6091, popularly known as the fading-numbers record. The patient replies not by writing but by speaking the numbers he thinks he hears.

By use of this record the child hears words with which he is thoroughly familiar. This record played on an ordinary gramophone cannot be used to test patients who have more than 30 db loss of hearing. As many of our patients are more deaf than this, amplification must be used. Our high-fidelity machine enables us to use this record to test patients with a loss of as much as 84 db. It has excellent attenuators, but we believe that the use of these in measuring hearing is inferior to the use of attenuation done in the recording.

We are convinced that a more useful estimation of hearing can be obtained by testing a young child with speech than by testing him with pure-tones. We are satisfied that numbers are superior to common words and sentences. Nonsense sentences and meaningless speech sounds we have altogether discarded.

Things We Do Not Perform in Testing Children's Hearing.

We do not use any test which cannot be performed in a very few minutes and be repeated exactly many times on different occasions; any test which does not command the child's interest frightens it, does not arouse its cooperation, or fatigues it. We have no use for watch tests, whisper tests, tuning fork tests or the monochord.

We do not use speech audiometry to check the validity and reliability of other hearing tests. Speech reception thresholds and discrimination scores have no value whatever in the testing of children's hearing. Diplacusis measurements, tinnitus matching, recruitment evaluation, loudness balance, pure-tone averages and practically every other test to be found on audiological work-up charts are useless in the testing of children.

Older, very intelligent children sometimes require modification of our regular testing technique, as also do young mental defectives. The amount of masking varies. These modifications are not stereotyped but are made on the spot by the otologist. The latter is qualified to do so, whereas an audiologist would not be.

What We Aim for:

Our aims are: an accurate and authoritative measurement of the child's hearing and a determination of whether his deafness is reversible or irreversible; therefore, we endeavor to use tests which we can repeat exactly, and also to use only those tests which we believe give us practical information.

Condemnation of Loud-Speaker Free-Field Testing.

Except in special cases, we advise against the use of loud-speaker free-field testing in any hospital clinic devoted to the hard-of-hearing — and especially in such a clinic for children.

This advice will cause amazement, because this scheme is the predominant feature of nearly every such establishment set up in recent years.

A hospital clinic is not a research unit primarily. A hospital clinic exists for the good of the patients and the otologists who serve them. A hospital clinic requires facilities for diagnosis and for comparative retests.

Nearly every technique in use is inaccurate to a degree — but this technique is much more inaccurate than most — and should not be used for these purposes.

The first important fault is that it makes use of live voice. Speech is influenced by the breath stream from the lower respiratory tract, modulated by the vocal cords, varied by the articulators (lips, jaw, tongue and soft palate) and its overtones are selected and suppressed by the vocal cavities (nasal, oral and pharyngeal). The whole process is governed by the brain. It is obvious that these basic speech processes cannot produce the same sounds for a second testing of the hearing for measured comparison unless they are in the same physical condition and are used in exactly the same way. Even if a very practiced technician can, with the help of a voice-level-meter, learn how to control the intensity of his voice to an error as low as 3 db, that does not make it possible for him to make his tests constant in intensity for more than the same day; nor does it prevent him, six months later, from having got out of his bed on the wrong side, or from an intimate sorrow upsetting his laryngeal control, etc. With such things to contend with, his testing procedure, six months hence, may not be the same as it is today.

That this contention is valid can be demonstrated visibly to anyone who visits the Western Electric research unit.

There can be seen in operation a large speech translator which throws speech on a moving phosphorescent plastic screen so perfectly that observers can read it. The speaker uses a voice-level-meter to help him maintain as even an intensity of speech as he can.

The visible speech patterns vary not only with the intensity but also with the pitch and with the timbre.

The effect of these on the visible patterns of speech can be quickly discerned by a study of the illustrations in "Visible Speech."[5] There, too, can be seen the variation in the visualization of the same sentences and the same words. It will be apparent that the sound pattern of speech continually changes.

The next disturbing fact is that all words spoken naturally and in sequence do not have the same physical power. In addition, even when carefully monitored to the same effort of speech, all words are not equally intelligible.

There are other difficulties in loud-speaker free-field testing. The acoustic properties of the soundproof room in which the loud-speaker is used are amazingly difficult to keep constant for subsequent testing: everything in the room must be exactly the same as was used at the previous test and in exactly the same position,—the patient must wear the same clothes; if not, the Western Electric Company has proven that the tests are not comparable.

The obvious conclusion is that live-voice testing for *measurement* is a delusion.

When one contemplates the many thousands of dollars which have been expended in such units and in paying the salaries of the required personnel, one wishes for the tongue and pen of a Jonathan Swift.

Necessity for Recorded Testing Material.

The consequence of this, namely, that live-voice must not be used, is that the testing material should be recorded. The present materials are phonograph record and sound tape.

Both phonograph records and sound tape are subject to wear and tear. They cannot be expected to retain their fidelity over an extended period of time; but the "master" can be expected to keep its accuracy for years and from it fresh records can be stamped *ad infinitum*.

Many recordings are at present available on various materials. Your clinic, your hospital, your office is under the

necessity of choosing one of these and laying in such a stock of them that your patients for many years to come may be tested by the same testing material under identical conditions.

All of the recordings I have studied have defects, but I am sure there are many I have not seen or read about. I am confident, however, that the clinical testing of the future will not be done by live-voice but by recorded speech. This may be phonograph record or tape record. The ultimate standard of either of these may not be established for some years to come. The point, however, is that the testing material will be a recording of some sort, so that it may be reproduced many times accurately. The tests done by such a method will be practical and the results will be comparable.

At present, in spite of its defects, I use in my clinic, Western Electric recording K.S. 6091.* This is popularly known as the fading-numbers recording. (I find the later record, Western Electric K.S. 10087, less desirable.)

It is the opinion of Hallowell Davis[7] (p. 140) that the use of the list of numbers in the Western Electric fading-numbers recording, properly administered, is a measure of the hearing loss for speech of the same precision and reliability as the pure-tone test of a clinical audiometer.

Criticisms of Our Scheme.

Having criticized other methods of testing hearing, it is proper to note the imperfections of the schemes I advocate.

The faults of the tape recorder are as yet unknown and will not be properly known until it has been in use by otologists as long as gramophone records have been used.

The audiologist criticizes the fading-numbers test thus. The recording can be obtained only on the old hard type of recording surface, which produces objectionable noises; both available recordings have certain imperfections which considerably annoy the tester; the relatively few combinations of the digits make the use of the fading-numbers record most

*The attenuation in the Western Electric K.S. 6091 is in decibel notation.

difficult to use in rehabilitative work because the short test lists can be memorized; it gives little or no information about discrimination.

The answer to these objections originates in the totally different viewpoints of the audiologist and the otologist. The latter is a physician; the former is not. The latter is interested primarily in his patient's satisfaction and well-being; the former is academic and interested primarily not in clinical research but in scientific research.

The surface noises, providing they are minimal, the loss of fidelity from wear, providing replacements are readily obtainable, the imperfections in the recordings, the memorizability of the lists, and the testing of discrimination are venial faults. They are all capable of improvement by alteration. They are as nothing compared with the value the records possess of being useful for comparative retesting.

It matters nothing to the audiologist whether a patient's hearing for speech improves, remains stationary or becomes worse, but to the patient and to the otologist these mean everything.

In some states, in the screening of the school population the fading-numbers test is being replaced by a pure-tone audiometer test because of certain supposed imperfections of the fading-numbers test. These are: loss of validity because the children, thinking it is some sort of school examination, are very apt to cheat or try to copy from each other; the method fails to detect early high tone deafness; the diagnostic curves show a lesser degree of accuracy than those of other testing methods; the operation of the test usually disrupts the entire class and too many repeats and referrals are required.

The advantages of the pure-tone audiometer for screening (which I personally doubt) are said by the protagonists to be: greater ease of operation; it is possible to command individual attention without interference from other children; the diagnostic curve shows greater degree of accuracy; it enables

a trained technician to examine an entire class with much greater speed than is possible with other methods and 20 to 30 children can be tested in both ears within an hour by sweep testing.

It matters not to us as otologists which of these methods seems to prove most practical for screening in schools. Our interest is entirely that children with incipient hearing impairment should be discovered so that appropriate steps may be taken early for their improvement. I will be glad to see the old screening method disappear from the public schools because the faults found with the fading-numbers test in school screening have been responsible for the lack of recognition of its very real merits.

The otologist is keenly interested in every test which brings him information which he can use for his patients' improvement. The great mass of the tests now used in audiology are of no use to him. His crying need at present is for a method which will provide data which can be compared with re-examination data of patients as they return. As I have inferred above, I believe that he can obtain this by using Western Electric K.S. 6091 in a high-fidelity reproducing unit.

The Hearing-Aid Problem.

A hospital hearing clinic may be asked to choose, to recommend and to evaluate hearing-aids; however, it is debatable whether any hospital should undertake this. Because of its commercial significance such work is surrounded by pitfalls which will not be enumerated here. In my opinion a great hospital, and much more a great university, should have nothing to do with it.

Every large city should endeavor to obtain unbiased hearing-aid service for the ever increasing multitude of hearing-aid wearers. In New York City such work is performed admirably by the New York League for the Hard-of-Hearing, a partially voluntary organization. In Great Britain such service is obtainable in innumerable cities and towns.

There are many hard-of-hearing children who are benefited by hearing-aids, and undoubtedly there are many more who could be. Every large city should provide classes designed to prepare children to wear hearing-aids. Until voluntary organizations or boards of education provide these, a children's hospital may be required to make provision to do this. It is, however, service which requires much space and special teachers, and it is debatable whether a hospital is the proper place for such work.

Need for a Teacher of the Deaf.

It is, however, certain that a hospital for sick children should employ an experienced teacher of deaf children.

It is commonplace that very few mature adults attain proficiency in lipreading, but those who work with little children know that the child of four or five learns to lipread very easily if he is intelligent.

It is the growing recognition of this that has led to the institution of preschool classes for the hard-of-hearing. In Toronto we have only one such class. Hence to a hospital for sick children will come mothers of children of preschool and younger age, not only from the city but from nearby municipalities and from all over the province. We hope to encourage such a mother to stay in Toronto for a long enough period to obtain instruction from our teacher on how to conduct herself with her child at home. Many an intelligent mother will learn to teach her little child to lipread with immense benefits to the child.

I believe that a well-trained, well-educated teacher of the deaf is indispensable for a hard-of-hearing clinic for children. It is all important that the one chosen should understand and love little children. She has to demonstrate patiently to the children and to the parents the fundamentals of how remnants of hearing can be utilized in the future education of the child, for it is the very early years of a child's life that are all important. The sphere of influence of such a teacher is very great.

Regarding Permanency or Irreversibility of Perceptive Lesions.

It is service to the little hard-of-hearing child that is the all important object of testing and examining it. The purpose should not be to decide whether or not an operation can be usefully undertaken by a fenestrationist. Consequently the location of the underlying lesion in the perceptive apparatus is not of prime importance.

As a heritage from the past, otologists make their first diagnostic objective the determination whether the lesion responsible for the deafness is or is not in the perceptive apparatus. Because there are obviously irreparable lesions of the internal ear, the assumption is made that every lesion of the perceptive apparatus is irreversible and, therefore, incurable. Having made such a diagnosis, any attempt to ameliorate the condition by treatment is considered futile and even dishonest.

We ought to know better. There are many lesions of nerves which recover. There are lesions of the optic nerve which recover. In disseminated sclerosis a degree of recovery may occur spontaneously and persist for many years. A large scotoma may diminish in size until it is almost completely gone. Multiple sclerosis and pernicious anemia are the two most common examples of demyelinating diseases where recovery can occur providing the axis cylinders have not been damaged to an irreversible degree. Hyland, Watts and Farquharson[8] in a long study of pernicious anemia patients have reported improved functioning of reversibly affected fibres in the tracts of the spinal cord due to maintenance of adequate therapy.

It is, therefore, safer to assume that lesions of the perceptive apparatus of the ear are, in their early stages at least, reversible and amenable to treatment.

Let me give you an example: a six-month-old patient of ours was a candidate for adoption. There was no evidence of any hearing and no ocular response to douching with cold water. We advised against adoption, and as our findings

remained unchanged we continued to do so. At 18 months, because its attempts at babbling were not flat and toneless, we said there was still hope. At the age of two years and one month the child commenced to put three and four words together.

We are in the infancy of an understanding of the problems of perceptive deafness.

We must be very slow in saying that a child who apparently has a serious perceptive lesion cannot recover.

As a corollary to the above assumption it is commonly believed that a lesion of the conduction apparatus is usually amenable to treatment. It is, however, easy to find in the literature pictures of fully organized deposits in the middle ear which are irreversible and incurable.

We should, therefore, stop concentrating on lesions of the internal ear and instead we should devote our attention to finding variations in the patient's hearing. Variation can occur with both conductive and perceptive lesions. If the hearing really varies, the underlying lesion is not yet permanently fixed and should be amenable to treatment. At present there is no test which demonstrates variability as effectively as the speech test.

Attention to Treatment Essential.

Children have immense powers of recovery and the otologist who finds variation in a child's hearing should be medically minded and not surgically minded.

The number of patients whose hearing is defective because of a congenital lesion is small. The vast majority are hard-of-hearing because of disease, usually infection. It is of fundamental importance that the otologist recognize the earliest signs of the disease and arrest the process at that stage if he can. He must watch the progress of the patient by measuring the hearing of the young child, a procedure which this paper has shown to be very difficult.

One otologist, whom I admire, has written that the testing of the hard-of-hearing children and their future handling should be the audiologist's task. It is my opinion that the audiologist will never be fitted to do such a task.

Visit any clinic you will, and you will be impressed by two things: the time devoted to audiologic technique and work-up is very great; much attention is given to the education of the parents by the audiologist; but the time devoted to history taking, examination, and treatment of the child is brief.

This is not as it should be. The most important part of all and the part that *should* be great is the estimation of the child's hearing ability by the otologist and his continued attention to the problem.

The measurement of the hearing in the very young child is extremely difficult and requires suitable techniques, but it is of fundamental importance because it is the indispensable index to the results of treatment.

Deficiency in hearing is caused in a small percentage of the population by defects present at birth. In the vast majority of instances, however, it is caused by disease, which itself has a beginning.

The defect in the hearing of an adult in many instances has been present so long that it is irreversible. The adult's defect should have been arrested at its beginning in childhood.

The treatment of suppurating ears in children should not be passed over to a nurse or young doctor, for the younger the child the harder the problem of treatment. It requires the care and perseverance that only an older otologist who is impressed with the responsibility of his task can give.

It is the ambition of this clinic of the Hospital for Sick Children of Toronto so to arouse the medical profession that early attacks upon the hearing apparatus of the young child will be arrested and the changes that have been produced will be wiped out while they are still reversible.

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BILATERAL MIDLINE PARALYSIS OF THE LARYNX OF CENTRAL ORIGIN.

ITS OCCURRENCE AS A POSTOPERATIVE COMPLICATION.*

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Bilateral midline paralysis of the larynx following surgery on a part of the body other than the thyroid is alarming because it is unexpected. It is serious because of the pulmonary complications which follow.

The author first observed a patient with such a paralysis in 1946. The patient, following section of the trigeminal nerve for tic douloureux, developed dyspnea, stridor and occasional cyanosis, on the afternoon of surgery. Direct examination of the larynx revealed the vocal cords immobile in the midline. A search of the literature at that time showed the only reference to such a postoperative complication to be made by Jackson.[1] He stated that bilateral incomplete paralysis of the larynx had been observed as a transitory condition after operations in regions remote from the thyroid—in two instances following abdominal surgery. It was assumed that the paralysis was caused by intracranial disturbances, probably circulatory.

Interest in the condition has been reawakened by our encountering three additional cases during the past two years.

Etiology — The sudden appearance of the paralysis and the bilateral involvement led to the conclusion that the causative lesion is a vascular one located in the medulla. The possible lesions are spasm, hemorrhage, thrombosis and embolism.

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Behrend and Riggs[2] state that cerebral complications following surgical operations are not rare, and found such complications were seldom the result of embolism or hemorrhage.

New and Childrey,[3] in a study of 217 cases of paralysis of the vocal cords, found 24 which were central in origin. Four were due to hematomyelia and one to thrombosis of the medulla. Three of the 24 cases were bilateral abductor paralysis.

Work[4] described a patient who presented clinical indications of multiple, small cerebral thrombi. Bilateral abductor paresis of the vocal cord was noted. Examined at a later date, the patient showed complete recovery.

Cody[5] examined a patient with sudden onset of paralysis of half the soft palate, half the tongue and abductor paralysis of one vocal cord. Re-examination seven hours later showed that all the structures were no longer paralyzed. He attributed the transient paralyses to vasospasm in the medulla. He enumerates thrombosis of the arteries supplying the medulla and vasoconstriction of the radicular arteries to the motor nuclei as vascular conditions causing the laryngeal syndrome of the medulla.

King and Gregg[6] list cerebral hemorrhage as a cause of laryngeal paralysis.

Location of the Lesion — Bilateral midline paralysis of the vocal cords is produced by involvement of the abductor fibres in the inferior laryngeal nerve of both sides. Corticobulbar fibres carrying impulses to the lower motor neurons, which supply the larynx, arise in the pyramidal cells in the precentral gyrus of the cerebral cortex. They pass downward through the internal capsule to the medulla, where they undergo partial decussation and terminate in the nucleus ambiguus around cells of the recurrent and superior laryngeal nerves; thus, impulses brought to the cells of origin of the recurrent nerve on one side arise from the cerebral cortex of both sides.

Furstenberg[7] points out that a lesion above the nucleus ambiguus will produce layrngeal paralysis only if large enough to affect the corticobulbar fibres on both sides. The paralysis would then be bilateral and spastic.

Jackson[1] points out that at, or near, the partial decussation of the motor fibres, a small lesion can reach the upper motor neurons of both sides and thus produce a bilateral motor paralysis of the larynx. Such a lesion, being above the nuclei ambigui, would be spastic in nature. Work[4] believes that spastic paralyses of supranuclear origin are often missed, especially if transient and incomplete. Furstenberg[7] believes that unilateral lesions are overlooked because of the slight impairment of laryngeal function, but that bilateral paralysis causes such a profound disturbance that a laryngologist is promptly consulted.

In the medulla, the nuclei ambigui are not far apart and the same is true of the recurrent laryngeal nerves; hence, a lesion here may cause a bilateral laryngeal paralysis. Such a lesion produces a flaccid paralysis.

Cody[5] points out that the radicular arteries supplying the nuclei ambigui are long and slender and more exposed to vascular disorders. They are more frequently affected than other vessels of the medulla.

Woodward[8] points out the close association of the nuclei of the glossopharyngeal, spinal accessory and hypoglossal with the vagus nucleus in the medulla and the resulting frequent association of paralyses of these nerves when the vagus is involved.

Cody[5] and Furstenberg[7] also stress the fact that central lesions causing laryngeal paralysis frequently involve the IXth, XIth and XIIth nerves in addition.

King[6] states that he has seen no laryngeal paralysis resulting from a central lesion that was not associated with paralytic disturbances of the muscles of the pharynx and tongue, and frequently with motor disturbances remote from the larynx; however, it must be remembered that vascular lesions sometimes cause transitory paralyses and the finding of associated paralyses will depend upon the relationship of the time of examination to the time of onset of the lesion.

Pathology and Pathologic Physiology—Behrend and Riggs[2] studied the protocol material from 21 patients who had cerebral complications following surgical operation. Five of these patients had shown symptoms of focal paralysis. In every case the brain showed generalized capillary damage with widespread parenchymatous degeneration due to pericellular and interstitial edema. In eight patients, who had survived the operation from two to nine days, there were focal areas of glial and phagocytic activity suggesting beginning repair and scar formation. Edema and congestion of the viscera were also observed. The anesthetic had been ether in 12 cases, cyclopropane in four, nitrous oxide in three, and procaine hydrochloride for spinal anesthesia in two.

These authors felt that the cerebral complications following surgical operation were due to cerebral anoxia resulting from acute circulatory collapse, precipitated by the administration of an anesthetic in persons with reduced circulatory reserve.

Wilson, Rupp, Riggs and Wilson[9] studied 542 cases of cerebral vascular accident. Their findings suggested that systemic circulatory inadequacy played an important rôle in the causation of cerebral vascular accidents.

Hicks and Warren[10] state that the cause of virtually all cerebral vascular accidents (excluding aneurysm) is local ischemia. This is true whether the lesion is hemorrhagic or nonhemorrhagic and whether or not thrombosis is present. In a study of 100 cases of fatal cerebral infarction, a majority of cases showed no evidence of mechanical occlusion of cerebral vessels. They conclude that vasospasm is the most common cause of the ischemia.

Sherman and Grinker[11] stress the relationship of cerebrovascular accidents to diseases of the cardiovascular system. They point out that, although there is a free capillary anastomosis within the brain, it is insufficient to maintain an adequate circulation in an area of any one artery which is occluded. Although the evidence is not entirely convincing that cerebral vasospasm of sufficient intensity to occlude an

artery can occur, they feel that, from a clinical standpoint, many cases of transient neurologic dysfunction are explicable only on such a basis.

Occlusion of a cerebral vessel for as long as two minutes results in a parenchymatous reaction with interstitial edema, hyperemia of neighboring vessels and reversible damage to brain tissue. The clinical signs of such a lesion may disappear within several hours or several days. Longer periods of occlusion produce irreversible damage.

Pickering[12] credits Peabody, in 1891, with being the first to ascribe transient attacks of paralysis to spasm of a cerebral artery. According to this theory, a cerebral arterial wall contracts, obliterating the lumen, the corresponding area of brain becomes ischemic and a corresponding localized sensory or motor paralysis ensues. Pickering feels the evidence for vasospasm of cerebral arteries is slight. He concludes that attacks of localized sensory or motor paralysis are due to edema of the brain. The edema is less likely to be due to constriction of the artery than to defective constriction.

Houssay[13] states that CO_2 is the most powerful dilator of the cerebral vessels and that ether also acts locally on the cerebral vessels to cause dilatation. Nervous disturbances of short duration have been attributed both to vasospasm and vasodilatation.

From a review of the foregoing extracts, we find that various authors agree that the basic cause of paralysis of central origin is local ischemia secondary to edema; that, if the paralysis is transitory, the local pathological lesion must be a reversible one; that long periods of ischemia cause irreversible damage to brain tissue and the resulting paralysis is of longer duration.

There is, however, wide divergence of opinion regarding the basic pathologic physiology. Some investigators feel that vasospasm is the primary cause. Others attribute ischemia and edema to vasoparalysis and circulatory insufficiency asso-

ciated with disorders of the cardiovascular system. In rare instances it may be due to hemorrhage, thrombosis or embolism.

Symptoms — As Jackson[14] points out, bilateral laryngeal paralysis is often overlooked. This should be especially true in patients recovering from surgery who are on bed rest and sedation. The outstanding symptoms of midline paralysis are inspiratory dyspnea and stridor. The symptoms vary in degree and may appear only on exertion. Usually the voice changes are slight.

Complications — With the obstruction to pulmonary drainage caused by the immobile vocal cords, secretional obstruction soon becomes prominent. In addition, if there is accompanying motor or sensory paralysis of the pharynx, the nasopharyngeal and salivary secretions find their way into the tracheobronchial tree to be added to the pulmonary secretions. Gray,[15] Von Leden,[16] Galloway[17] and others have called attention to the seriousness of the accumulation of these secretions in the lower bronchial tree.

With obstruction to the removal of secretions, reduction in the ventilation capacity occurs. This leads to hypoventilation, retention of CO_2 in the body and an increase of CO_2 in the blood. Hypoventilation also reduces the O_2 saturation of the arterial blood. This disarrangement of the relative O_2 and CO_2 content of the blood soon brings about a change in blood pH. A combination of these factors leads to asphyxia.

In the early stages of such complications, patients show restlessness, apprehension and disorientation. This may lead to further sedation and further depression of the respiratory center. Two significant early signs are nonproductive cough and the presence of rhonchi in the chest.

Usually at this stage the patient is treated by postural drainage, catheter suction and perhaps bronchoscopic aspiration. In addition the patient receives O_2 therapy. All of these measures fail to relieve the dyspnea and stridor for any significant length of time.

If the obstruction continues, edema, atelectasis and pneumonitis appear.

Diagnosis — If the laryngologist is consulted, mirror examination shows the cords to be immobile in the midline. Therapeutic bronchoscopy for the aspiration of accumulated secretions may also reveal the diagnosis; however, unless the possibility is kept in mind, the endoscopist, in his anxiety to relieve a very sick patient, may overlook the paralysis since the bronchoscope is passed without too much difficulty into the trachea. The paralysis viewed through the bronchoscope may be mistaken for a spasm of the glottis.

The inspiratory stridor and failure of aspiration of retained secretions to relieve the dyspnea should suggest obstruction in the larynx itself.

Differential Diagnosis — The important differential diagnosis in postoperative laryngeal paralysis is to differentiate it from obstruction that is due to simple retention of retained secretions. This is important because, while aspiration of secretions, postural drainage and administration of O_2 may suffice in the treatment of the latter condition, they will prove insufficient if the vocal cords are paralyzed.

Abductor paralysis must also be differentiated from postoperative edema of the mucosa of the larynx and of the hypopharynx. Modlin,[18] in discussing a paper by Gius and Grier, reported six patients requiring tracheotomy postoperatively following bilateral neck dissection with removal of both internal jugular veins. This procedure was necessitated by alarming postoperative edema of the larynx. It was necessary to maintain the cannula in place for as long as three weeks postoperatively. In one of our cases of laryngeal paralysis following bilateral neck dissection, the paralysis was not apparent until the edema of the hypopharynx and larynx had begun to subside.

At times the reaction to an intratracheal tube used for anesthesia may confuse the diagnosis. This was true in another of our cases which showed moderate ecchymosis and edema of the pharyngeal mucosa in addition to the laryngeal paralysis.

During an epidemic of poliomyelitis, particularly following an emergency operation, it would be well to differentiate the bulbar type of polioencephalitis from abductor laryngeal paralysis.

Treatment — The immediate treatment of bilateral midline paralysis of the vocal cords is tracheotomy. The introduction of a tube into the trachea bypasses the laryngeal obstruction and in cases of associated paralysis of the pharynx, either motor or sensory, it lessens the danger of aspiration of secretions. If the complication of secretional obstruction has already supervened, this condition is more readily treated by repeated aspiration through the tracheotomy cannula.

The importance of treating the primary condition is emphasized by the findings of Ende and Ziskind,[19] who in a series of 137 consecutive autopsies found three postoperative deaths due to asphyxia from retained tracheobronchial secretions.

Fortunately, the present day tendency is to treat all cases of respiratory difficulty due to retained secretions by tracheotomy if the condition is likely to persist for more than 24 hours. This makes for ease of nursing care by personnel with no specialized training. Thus, even if the diagnosis of a postoperative laryngeal paralysis is missed or delayed, the proper treatment would possibly be instituted on an empiric basis.

The majority of cases of laryngeal paralyses that we have seen complicating surgery other than thyroidectomy, have resolved in from four to six weeks; however, if the paralysis persists longer than six months, it is likely that gliosis and scar tissue formation have taken place in the central area involved. In such a case surgery on the larynx as devised by King, Kelly, Woodman and others may be necessary if the patient is to be decannulated.

CASE REPORTS.

Case 1: A female patient, past the fifth decade, was admitted to a voluntary hospital in Albany, N. Y., in January, 1946, because of trigeminal neuralgia. Under general intratracheal anesthesia, a section of the Vth nerve was performed intracranially. Shortly after recovery from the

anesthetic, the patient had an attack of cyanosis. The anesthetist was called and he sspirated the tracheal mucosa and administered oxygen. Because the patient had anesthesia of the pharynx, it was possible to examine the vocal cords directly. This examination revealed a bilateral midline paralysis of the vocal cords. Because the dyspnea persisted with recurrent cyanosis, a tracheotomy was performed on the same evening as the original surgery. This relieved the patient's symptoms. During the next few days, the anesthesia of the pharynx disappeared and the mobility of the vocal cords returned within two weeks. The patient was then decannulated, the tracheotomy wound closed by a plastic procedure and the patient discharged from the hospital.

Case 2: E. H., aged 66, white female, was admitted to the Veterans Hospital for surgery of a recurrent carcinoma of the breast. Original surgery on the breast had been performed in February, 1946. and followed in 1948 by irradiation. The patient had a systolic murmur in the mitral area but there was no cardiac enlargement. On Oct. 16, 1950, under pentothal and intratracheal nitrous oxide anesthesia, the lesion of the chest wall was removed and a skin graft applied. On the second postoperative day, the patient was examined because of dyspnea and stridor. There was ecchymosis of the palate and swelling of the false cords. The true cords were not visualized. Chest X-ray showed no atelectasis. Two days later, because of persistent dyspnea, a direct laryngoscopy was performed and it was then found that the patient had a bilattral midline paralysis of the flaccid type. Tracheotomy was performed, which relieved the dyspnea. By Nov. 15, 1950, the cords were moving normally on abduction and the tracheotomy tube was removed.

Case 3: C. H., aged 63, white male, was admitted to the Veterans Hospital on Sept. 20, 1951, because of pain in the abdomen of one week's duration. In 1945, the patient had suffered a compound fracture of the skull with damage to brain tissue. This left him with a left hemiparesis. Chest X-ray on admission revealed moderate cardiac enlargement. Abdominal examination revealed an incarcerated mass in the right inguinal region. His blood pressure was 128/70. An emergency inguinal herniorrhaphy and appendectomy were performed on the day of admission under spinal anesthesia. Late on the day of surgery, a bronchoscopy was performed because of signs of retained secretions. Two days postoperatively, a chest X-ray showed pneumonitis at the left base. On the fourth day, laparotomy was performed because of ileus. Bronchoscopy was repeated at this time. Chest X-ray then showed atelectasis. In spite of the administration of oxygen, the patient was dyspneic and cyanotic and moist rales were present throughout the chest. The patient was confused and disoriented and for this reason was receiving sedation. He had also been receiving antibiotics with no appreciable effect upon the chest condition. He was coughing but failing to raise any secretion. On Sept. 28, 1951, he was seen in consultation by the laryngologist. Examination revealed weakness of the tongue and palate and a bilateral midline paralysis of the vocal cords. Tracheotomy was performed and a large amount of thick mucopus aspirated from the tracheobronchial tree. The patient's condition improved remarkably within 24 hours, both physically and mentally. Five days later, the patient was able to swallow nourishment. Within three weeks the paralysis of the vocal cords had disappeared. The tracheotomy tube was removed and on Oct. 17, 1951, the wound was closed by a plastic procedure. The patient was discharged to the domiciliary section on Jan. 7, 1952.

Case 4: R. W., aged 68, white male, was admitted to Veterans Hospital on Aug. 6, 1951, because of a mass in the right cervical region. This mass

had been present for seven months. Ear, nose and throat examination revealed an ulceration at the base of the tongue on the right, extending past the midline. Another cervical node was noted low in the left cervical region. His blood pressure was 156/78. Physical examination and laboratory examinations revealed no other abnormalities. Biopsy of the tongue ulcer showed squamous carcinoma, Grade II. Deep X-ray therapy showed that the ulcer responded to irradiation. On Sept. 13, 1951, seven radon seeds of 2.5 millicuries each were inserted in the tongue and on the following day, under intratracheal anesthesia, a right radical neck dissection was performed. There was moderate postoperative edema of the pharynx and right aryepiglottic fold; however, this edema and the irradiation reaction of the tongue gradually subsided. The incision healed slowly because of the irradiation. On Nov. 13, 1951, under intratracheal anesthesia with nitrous oxide and ether, a left radical neck dissection was performed. When the intratracheal tube was removed at the conclusion of the operation, the patient became cyanotic. It was impossible to reintroduce the tube because of the pharyngeal and laryngeal edema. A tracheotomy was performed. This relieved the cyanosis. Postoperatively, there was marked edema of the conjunctiva, pharynx, larynx and cervical region. This gradually subsided. Mirror examination of the larynx was made on Dec. 7, 1951, because of inspirational stridor and because patient had dyspnea when the tracheotomy tube was occluded. This revealed a bilateral midline paralysis of the vocal cords. Occasionally during the next two months the cords seemed to regain slight mobility; however, five months postoperatively, with all edema of the face, conjunctiva and larynx subsided, the cords lie immobile in the midline, and the patient can go only a short time with the tube occluded. He also has much difficulty in raising mucus unless the tracheotomy tube is open. Surgery for bilateral abductor paralysis of the vocal cords is now being contemplated.

DISCUSSION.

In Case 1 an associated paralysis of the glossopharyngeal nerve was noted when the patient was examined shortly after the onset of symptoms. In Case 3 a paralysis of the hypoglossal nerve was also present. No associated paralysis was noted in the other two cases, although such a condition may have existed temporarily.

Case 2 illustrates well the pulmonary sequelae of obstruction with retention of tracheobronchial secretions. It also shows the mental results of anoxia even when O_2 is being administered when the bronchial secretions are not being evacuated.

In Case 4 it was difficult to convince the general surgeon that the obstruction was not due to local edema of the larynx and pharynx; however, the persistence of the bilateral paralysis after all edema had subsided seems convincing proof that the paralysis was central in origin.

SUMMARY.

Four cases of bilateral midline paralysis of the larynx occurring as postoperative complications due to central vascular lesions are presented. Two cases showed associated paralyses. In three cass the paralysis was temporary. In the fourth case it has persisted over five months and appears to be permanent.

The pathophysiological possibilities are discussed.

Tracheotomy for the treatment of the paralysis and its pulmonary complications is emphasized.

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PERCEPTIVE DEAFNESS IN CHILDHOOD.*

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There appears to be very wide variation in the available figures on the incidence of deafness in children. Very roughly one may say that one in every 300 boys and one in every 400 girls are sufficiently deaf to require special schooling, and that of these the acquired, postnatal types have been approximately twice as common as the congenital. Since both varieties are frequently the result of epidemic disease, statistics must vary considerably from time to time, and from place to place; also, due to constantly improving public health services and epidemic control, acquired forms of deafness should, in time, be very largely eliminated. Due to lack of precise knowledge, classifications of nerve deafness have mostly been unsatisfactory. The table used in this presentation is similar in most respects to that recently suggested by Victor Goodhill.

1. *HEREDITARY* (GENETIC)

   A. Developmental Anomalies

      Aplastic
      Degenerative

   B. Erythroblastosis Fetalis (Kernicterus)

2. *ACQUIRED*

   A. Prenatal:

      1. Infections (Rubello — polio — viral)
      2. Drugs
      3. Developmental (Metabolic — Endocrine — etc.)

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B. Natal:

1. Birth Injuries
2. Drugs (Quinine)

C. Postnasal:

1. Local Infections (Labyrinthitis)
2. Systemic Infections (Esp. Viral)
3. Drugs (Streptomycin)
4. Trauma (Auditory and Gross)
5. Miscellaneous

1. Hereditary Deafness

A. Developmental —

Familial deafness is transmitted according to the Mendelian pattern, involving a single recessive gene. Its occurrence in the offspring of a deaf patient of this type depends upon the hereditary makeup of the spouse — thus the influence of consanguinity. The presence of this type of deafness among the siblings, even if the subject has normal hearing, suggests that he is a carrier of the recessive gene for deafness.

In developmental anomalies, there is either a primary lack of development or there is development up to a point followed by regressive changes. It consists essentially of one or more anomalies of the cochlear duct usually associated with abnormalities of the saccule and rarely of the bony cochlea, the utricle or semicircular canals. It is also often associated with other developmental anomalies such as feeblemindedness, retinitis pigmentosa, familial osseous fragility, and various bodily malformations.

Complete deafness is rare. In making the diagnosis, a clearcut history is essential in distinguishing this from other forms of deafness.

B. Kernicterus —

The second variety of hereditary deafness is of considerable interest at the present time. The mechanism is briefly as follows:

Persons are said to be Rh positive or Rh negative according to whether their blood contains Rh agglutinogen or not — 85 per cent of people being positive and 15 per cent being negative. Rh negative persons are liable to develop antibodies — *i.e.*, against Rh positive cells — if Rh agglutinogen is introduced into the circulation. This may occur in either sex as a result of transfusion, and in an Rh negative woman who is pregnant with an Rh positive fetus — the fetus having inherited the agglutinogen from the father. It appears that the agglutinogen of the fetus enters the maternal circulation, thereby stimulating the production of the specific antibodies. These in turn on entering the fetal circulation in sufficient concentration produce hemolysis. Three degrees of this condition have long been recognized, namely:

1. Congenital hydrops fetalis — the infant being either moribund or dead at birth.
2. Icterus neonatorum in which there is complete jaundice at or within 24 hours of birth.
3. Congenital anemia of the newborn.

Unless a woman has been sensitized by transfusion, the firstborn is likely to escape, due to lack of concentration of antibodies in the maternal blood; but the second, and still more the third, child is likely to be affected.

The cerebral cortex and cranial nuclei are the areas chiefly involved. Rh incompatibility occurs about once in 200 births.

2. Acquired Deafness

A. Prenatal —

1. Infections — Some years ago maternal rubella became suspect, since when its rôle as the major single cause of infantile deafness has been steadily unfolding. The cochlea develops from the sixth week until about the end of the fourth month. The degree of deafness depends largely upon the stage of development of the cochlea at the time of onset of

the infection. Maternal rubella has been found to be the cause of 20.5 per cent of all forms of infantile deafness, and this figure is likely to increase (Goodhill).

As might be expected from the nature of the condition, deafness from this cause is frequently associated with other defects and malformations, including: congenital heart disease, congenital cataract, harelip, club foot, microcephaly, hypospadias, spasticity, etc.

It is a curious reversal of form that the other exanthemata, which can play such havoc with the hearing of infants and young children, seem to have little or no effect upon the fetal organism; however, another virus disease which has been found to have an unexpectedly high incidence as a cause of deafness is poliomyelitis — again when it occurs during the first trimester.

2. Drugs — It is of interest to note that much experimental work has been done in animals on the effects of drugs on the fetal organs of hearing. Investigators have reported that changes in the cochlea can be produced which are indistinguishable from those found in developmental deafness. Some of the chemicals mentioned are calcium chloride, pilocarpine, guanidine, arsenic acid. These findings would lead one to think that deafness from intrauterine toxic factors of exogenous origin may be more common than is generally suspected.

B. Natal — Birth Injuries — Drugs (Quinine)

C. Postnatal —

Statistics indicate that this reaches a maximum during the first two years of life, after which there is a steady decline — becoming more rapid with each year achieved; however, from the point of view of therapy, there is nothing to choose between a young child who has been born deaf and one who has acquired his deafness within a year or two of birth. Neither one can hear, and because of this essential lack, they are both quite incapable of speech without having special training.

Infections — Inflammatory states in which the cochlea is invaded form a major cause of this type of deafness, and are generally the result of meningitis, the epidemic form being the greatest offender. The mechanism is that of direct spread of infection from the meninges to the cochlea and vestibule by way of the VIIIth nerve and the cochlear aqueduct. The contents of the bony labyrinth become replaced to a greater or less extent by the products of infection, which, with survival of the individual, become organized, and form granulations, and these in turn may complete the metaplastic cycle to become bone.

Meningitis alone appears to be the cause of approximately half as many cases of deafness as maternal rubella. It is closely followed by the virus infections — the exanthemata; however, it must be borne in mind that the majority of infections, both regional and systemic, are capable under suitable circumstances of causing deafness. This usually affects the cochlea and only occasionally the VIIIth nerve, for, since the cochlea is phylogenetically the most recently developed organ of special sense, it is correspondingly the least well able to resist or to recover from any form of injury.

Drugs — Many drugs have been implicated. One of recent import is streptomycin. Deafness from this cause is likely to be both permanent and severe. Recent investigations have shown that at least in some instances the lesion in streptomycin deafness is liquefactive necrosis in the ventral cochlear nuclei. The effect of streptomycin appears to differ from that of dihydrostreptomycin.

Trauma — Another small group, but one which is acquiring greater significance in this modern age, is that of deafness due to trauma. Briefly one can say that if the labyrinth is involved in the line of fracture, the resulting deafness will probably be complete, presuming that the patient recovers.

If the temporal bone is involved, but the labyrinth is not directly implicated, the degree and permanence of the ensuing deafness depends upon the amount of blood which invades the labyrinth and upon its subsequent absorption or organization.

Much the same picture may follow concussion without fracture, and at times from a seemingly trivial injury.

Age plays a definite part in this category in that the older the patient, the less hearing does he recover from his point of maximum deafness.

Diagnosis: As regards the *recognition* of deafness in children, it is interesting to note that it is more often the inability to speak than the inability to hear which is first recognized and for which cure is sought. So much is speech a function of hearing that without proper training, every child who loses his hearing prior to the age of four years will most certainly become a deafmute, and this is likely to occur with loss of hearing up to the age of eight or even 10 years. Another important sequel to the loss of hearing is that the older the child on suffering this loss, and the longer he remains without training, the more likely are personality problems to develop to the detriment of his rehabilitation. The psychology of the deaf is peculiar; it produces an exaggerated sensitivity about their affliction with a tendency to withdraw, to irritability and to suspicion.

In the very young the estimation of the degree of deafness is unquestionably difficult, but, as already indicated, it is more important to recognize at the earliest opportunity that there is deafness present than to name its type.

Tests — Testing is of two varieties:

1. The usual type in which the patient voluntarily responds to the test sounds and, —
2. Objective hearing tests in which the examiner is able to observe involuntary responses.

The latter is, of course, the kind which has to be employed for infants. The most accurate, but somewhat complicated, is probably Bordley's galvanic test in which the patient is conditioned to a faradic stimulus, the response being in the form of a galvanic skin reaction. Though less accurate, the much simpler and more frequently used method is to observe various normal, reflex reactions to sound — such as twitching of eyelids, face or limbs.

In eliciting these responses in an infant, a great variety of instruments has been used — bells, triangles, drums, etc., the percussion instruments supposedly being most suitable. Later, in an effort to gain a better idea of the range of hearing, variously pitched pipes, whistles, etc., are recommended. After the age of four or five years the audiometer may be tried, but the young tend to lose interest rapidly. Many investigators advocate the early use of the audiometer by means of play conditioning, *e.g.*, "the peep show," as suggested by Hallpike and associates.

Seldom is there a completely dead cochlea. If this seems to be the case, the static labyrinth should be investigated. Though occurring more frequently in postnatal than prenatal deafness, complete lack of vestibular response is most uncommon.

Differential Diagnosis: Since failure of speech development is the most outstanding feature of the severer forms of deafness, any condition which produces or is associated with this failure may cause confusion in the differential diagnosis.

The four principal conditions which should be kept in mind in this regard are:

1. Mental Retardation — in which there is a backwardness in nonauditory tests, visual inattention, emotional and intellectual lack of response, as opposed to the alertness of the deaf child.
2. Speech Delay from Emotional Cause or Lack of Incentive, *i.e.*, the child of overindulgent parents, or, at the other end of the scale, the institutionalized child.
3. Motor Speech Delay, which is often seen to coincide with efforts to train away from a preferred left-handedness.
4. Developmental Word Deafness — an interference with the receptor mechanism which makes speech unintelligible to the subject and, therefore, impossible of reproduction. Clinically and therapeutically he is an aphasic.

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REHABILITATION OF THE DEAFENED CHILD.*

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One of the most important basic needs of humanity is the ability of communication with one's fellow-men. In the normal chain of events, this faculty of communication depends to a great extent upon the two functions, hearing and speech; however, in the problem with which we are concerned at the moment, we have learned that it is possible for the added senses of touch, sight and the sense of movement to be utilized in place of, or in addition to, hearing.

For a moment, let us briefly review the history of education of the deafened. In Roman times, the deaf and mute were classed with the mentally incompetent, and this condition persisted almost without exception until the middle of the sixteenth century. Then Cardano of Padua stated that the deaf could be taught to comprehend written symbols or combinations of symbols by associating them with the object or the picture of the object they were intended to represent; that is, the association of meaningful language with experience. Closely following Cardano, Pedro Ponce de Leon of Italy and Bonet of France commenced the oral education of deafened children. In 1680, Dalgarno of Italy stressed the fact that deafened individuals were as mentally alert and capable of instruction as a normal person and "there might be special addresses made to a dumb child, even in his cradle." In the eighteenth century, the outstanding figures were Abbé Charles Michel de l'Epée in France — who favored signs, and Heiniche in Germany — advocating speech and speech reading. The influence of these men was worldwide. In 1817, the first permanent public school for the deaf in the United States was

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founded in Connecticut by Gallaudet. This was the forerunner of a great system of schools for the deaf, backed by the enthusiasm of Gallaudet's son, Sarah Fuller, Alexander Graham Bell, Caroline Yale and Dr. Max Goldstein. Thus through the work of these and many others — doctors, physicists, psychologists, sociologists and teachers, we have reached the present status, where all deafened children have the opportunity of education and communication with their fellow-men.

Any proper system of education for the deafened child depends upon four main factors:

1. Parental education.
2. Proper diagnosis and estimation of residual hearing.
3. Education by means of auditory training, language training, speech reading, and the use of the hearing aid where indicated.
4. An adequate follow-up program.

These factors must be considered in detail.

PARENTAL EDUCATION.

Many parents are loath to admit and accept the fact that their child will never hear. They squander much time and money on consultations in one center after another, not realizing the harm they are doing their child by not instituting measures as early as possible. Again, other parents accept their child's deafness, and then attempt to "make it up to him" by overdevotion — doing everything for him and shielding him from all contacts with normal living. Thus it is that when the child commences his program of education, not only has he been deprived of communication with others, but he is a maladjusted and thoroughly spoiled child as well.

The parents should be taken freely into the confidence of the otologist. They should be told why the child does not hear, and what can be done to help him, stressing always the important part that they will play in that education. If possible, they should be present at least at the first few lessons of the

teaching program, in order that they may work in close cooperation with the educator. The parents must be shown that education begins at the time the diagnosis is made or even presumed. If the child is still a baby, it is held on the lap so that it can see the parent's face and talked to, kindly, lovingly, and in a normal fashion, not with mouthings and grimaces. Then the baby is held against the face and the chest, talking constantly and repetitiously so that it may perceive vibratory stimulation. Gradually the child comes to associate the movement of the lips with the vibratory stimulation, and the training of the child has begun, in the mother's arms.

The Ewings of Manchester have found that the voice of a deaf child of one year is not noticeable by its abnormalities. There is rhythm to the voice and pitch and intensity can be varied, though the variations are fewer than normal; however, if left alone, about the age of 18 to 24 months, the deaf child tends to lose this range of vocal expression and the voice takes on a strained quality. Gestures replace vocalization. Once the deaf child ceases to use his voice, the natural intonation never can be recovered. Thus the use of the voice must be constantly encouraged, so that the sounds become automatic and eventually form the natural basis for the development of speech. Articulation has to be taught; speech as such can never be taught. It must become part of the child's mental and social experience and must be the result of an inner urge. All this lies within the realm of parental teaching, but they in turn must be helped greatly.

To facilitate the parents' education, various reading material may be recommended! the *Volta Review*, the publication of the American Association to Promote the Teaching of Speech to the Deaf, and the Volta Bureau; the *Hearing News* of the American Hearing Society; they may enroll in the John Tracy Correspondence Course; or they may contact their Provincial School for the Deaf for further educational material. In this way, the parents gain more insight into their child's problem.

PROPER DIAGNOSIS AND ESTIMATION OF RESIDUAL HEARING.

The matter of diagnosis has been covered very adequately in a preceding paper. It has been shown that practically all of the congenitally deaf cases have some island of residual hearing which can be utilized for educational purposes. Likewise, many of the cases of acquired deafness have some degree of hearing reserve. In a series of 460 cases investigated at the Pennsylvania School for the Deaf, residual hearing to one frequency was found to be present in 95 per cent of cases, but only 25 per cent could hear frequencies above 512 d.v.; however, any degree of residual hearing can be utilized in speech reading, where the child gathers his speech impressions from all his senses at the same time — visual, kinesthetic or muscular cues, and auditory.

The initial stage in the estimation of residual hearing is to gain the interest of the child. In most cases, there has been no preliminary parental education and the child is presented as a fretful, restless and ill-tempered being, with willfulness and self-determination the predominant emotions. No attention is paid to sound, and until the child is taught to listen, little progress can be made. To initiate this desire, training in a conditioned response is begun. The simplest conditioned response is that of raising the hand when a loud sound is presented at the ear. As a preliminary stage, this sound should stimulate both the auditory and tactile senses, such as a buzzer operating a telephone receiver. Thus, even if no hearing is present, with proper training a conditioned response to a tactile stimulus is set up; but as we have shown, 95 per cent of deafened children have some residual hearing, so we proceed from there. It is to be noted that throughout the training period, the child's behavior improves remarkably. He becomes attentive and obedient and easier to handle in all ways. When it is decided that the responses to the training device can be relied upon, a reasonably accurate audiogram may be obtained. We are then ready to proceed to the actual education of the child.

EDUCATION.

The education of a deafened child should proceed along the lines of auditory training, speech training and language, and speech reading.

First, auditory training. As has been intimated previously, if there is even a suspicion that a child is deafened, his early years should be filled with loud and varied sounds. Thus, he is taught awareness of sound which provides another link to the world about him. This awareness of sound leads to the next step of discrimination of sound, *i.e.*, the ability to differentiate one sound from another. For this purpose, the teacher may use a drum, a bell, a rattler, a whistle (preferably of low pitch) and a bicycle horn. The child is shown the objects and the noise they make. He is allowed to play with them and sound them himself. Then the sound is made out of sight of the child and he is required to pick out the instrument from which the sound emanated. From this stage, finer discriminations may be made; for example, bells of different pitch. Once a child learns that sounds differ from one another, he is ready to start learning to understand speech. Here again one begins with the gross discriminations between vowels — "ah" and "ee" — quite dissimilar. Then one works slowly on to the consonants. Following this, the child is taught to respond correctly to short sentences — "Bring me your coat," "Where is your hat?", etc. In this way the child assigns meaning to the phrase without analyzing each part. It closely follows the normal pattern of learning. As training progresses, finer discriminations are taught — the difference between "S" and "SH," between "TH" and "F." He must know and understand a large vocabulary of words, and must be able to follow connected speech from conversation periods, stories, phonograph records, etc.

A very important adjunct to auditory training is the hearing aid; however, before a child is fitted with a hearing aid, his auditory training must have progressed through the stage of awareness of sound and discrimination of sound to a degree that he will receive some practical benefit from the instru-

ment. Every acoustically handicapped child who can benefit from a hearing aid should be fitted with his own instrument as soon as he is ready for it. At first, the teacher covers her lips to forcibly submerge lipreading. Later lipreading and hearing are trained simultaneously. Only with the hearing of speech can the proper imitation of speech be possible, and the flat, unintelligible speech of the deaf be avoided.

In this connection there has been an instrument produced by the Maico Co., called the Train-Ear, which consists of amplifier, phonograph turntable and radio, which is proving of great value in the re-education of the hard-of-hearing child.

The second stage in education consists of speech learning and language. Now that the child has become aware of sound, and his residual hearing has been trained to interpret differences in sound, and the voice as a medium of communication, he must be taught to speak properly. In the child with a sufficient reserve of hearing in all frequencies, this is a matter of continuing the foregoing training, with emphasis on the hearing aid, and speech reading; however, in those cases in which there is residual hearing only for low frequencies or no residual hearing whatsoever, it becomes a more painstaking procedure, and additional methods must be utilized. In the development of language, it is necessary to associate an idea with a word. Once the recognition of this association is obtained, the development of language has begun. To teach the word "jump," the teacher jumps. To teach the word "ball," the object is shown, together with the name of the object on a card. Thus, gradually, a limited vocabulary is built up.

In building up a child's store of language, three major channels are substituted for the ear: vision, touch and muscular movements or kinesthetic clues. By visual clues, the child learns to interpret gestures and the facial activity that is the visible part of speech. He is then started on the road to speech reading. Through the sense of touch, the child learns of the plosive consonants (P. B, etc.) by feeling the gust of breath. By the hand on the cheek, the nose or the larynx, he gets further information on certain sounds which

give most vibration in these areas. By producing the same vibration in himself, he is continually adding to his vocal knowledge. By the kinesthetic cues, the child learns of the jaw movements, tongue movements, etc., necessary for the formation of intelligible speech. Thus, whereas the normal person judges speech on how it sounds, a deaf child must base his speech on how it feels.

The third important part of education consists of speech reading, a more inclusive term than lipreading. In the English language, over 50 per cent of all speech elements are either invisible or indistinguishable. In the normal flow of language the invidual speech elements have a duration of only one-thirteenth of a second; therefore, visual perception of the spoken language cannot depend upon the recognition of the individual letters, but must also take into consideration muscular movements, as well as context and other clues. The deafened child must observe the rhythm of sentences, the timing of syllables and the pauses between, the facial expressions and gestures. Thus, he must be continually on the alert, and in many cases, tension and fatigue are produced in a short time; however, it is from this final stage in his education that the child achieves the ability of communication with others.

The education of a deafened child is thus shown to be a continuous process from the time the deafness is diagnosed or presumed, through the stage of parental education, nursery school and specialized schools for the deaf until they are judged able to continue their studies with normally hearing children in public school. The earlier training is begun the sooner can this objective be obtained. It has been found that with preschool training 90 per cent of partially deafened children and 50 per cent of profoundly deafened children can carry on at public school; however, without this preschool training, only 49 per cent of partially deaf and 1 per cent of profoundly deaf children were able to carry on public school instructions.

The responsibility does not end here. There must be a comprehensive follow-up program. The hard-of-hearing child, who relies upon a hearing aid, must be rechecked regularly,

and, if his hearing is deteriorating, appropriate lines of training must be instituted. Some children may fall into slovenly habits of speech or their speech reading may deteriorate, necessitating a brief brush-up course; but, throughout it all, slow and painstaking as it may seem, is the constant inspiring thought that a child has been salvaged to lead a normal life and take his place in society.

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ODONTOGENIC TUMORS AND CYSTS.*†

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Odontogenic tumors and cysts are frequently encountered in the practice of otorhinolaryngology. Such encounters are often fraught with confusion, for several reasons.

The terminology used in discussing these structures is usually more familiar to the dentist than to the doctor of medicine. The nosology, *i.e.*, the classification, of these conditions is difficult. Some would include various developmental malformations, such as the fissural cysts.[1] This paper includes only those conditions purely odontogenic (Gr. odous or odont, tooth plus genneo, I produce or generate). Finally, as emphasized by Willis,[2] the understanding of these conditions is based upon an understanding of the embryology concerned.

GENERAL STRUCTURE OF A TOOTH.

These fundamentals, the terminology, nosology and embryology, must next be considered, first starting with the general structure of a tooth (see Fig. 1). As indicated, there is a crown and root portion, with enamel, dentine, cementum and pulp constituents. The embryological development of these components must next be considered, since it is their maldevelopment which accounts for the odontogenic tumors and cysts.

EMBRYOLOGY.

Starting about the fifth or sixth week of embryonic life, there occurs a thickening of the oral stratified squamous

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mucosa at various points along the alveolar process (see Fig. 2). Observing one of these thickened areas diagrammatically, and following it along, the proliferation is seen to continue

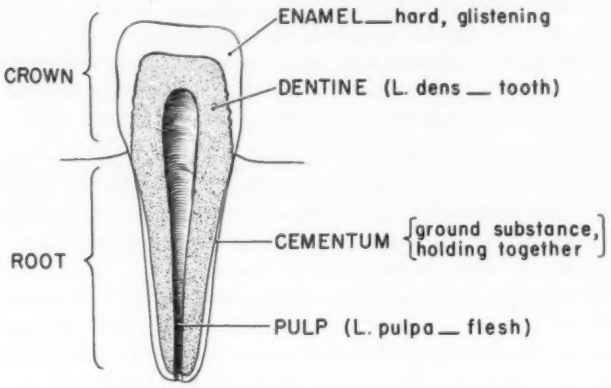

Fig. 1.

until there is a downward extension of the epithelium into the submucosa. The appearance of this downward projection characterizes the so-called "bud stage" (see Fig. 2).

As this bud extends deeper, a knob-like thickening occurs at its deepest portion (see Fig. 3). (A second thickened area at the middle of this bud will eventually form a permanent tooth.) This knob-like end then begins to flatten out, so that the entire structure looks somewhat like a mushroom. The stem of the mushroom, the cord of epithelium which extends from the oral surface into the submucosa, is called the dental lamina. The cap of the mushroom, for which reason this is designated the "cap" stage, consists of this flattened epithelial structure which now develops a central area of different consistency. This area is filled with a loose, stellate reticulum. Toward the oral mucosa the stellate reticulum is bordered by what is now designated the outer enamel epithelium; its other, deeper surface is bordered by the inner enamel epithelium. These three collectively are designated the enamel organ.

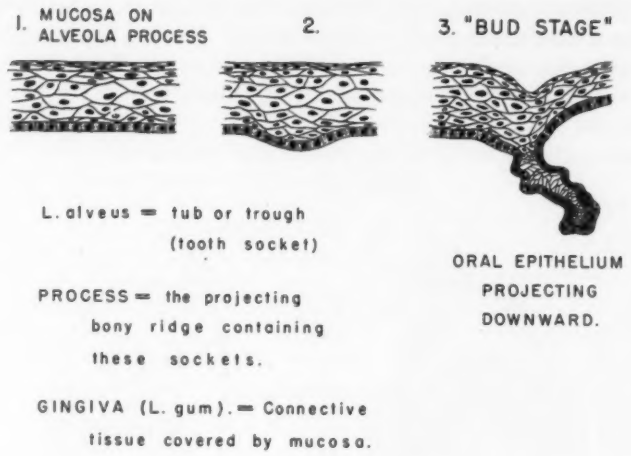
1. MUCOSA ON ALVEOLA PROCESS
2.
3. "BUD STAGE"
L. alveus = tub or trough (tooth socket)
PROCESS = the projecting bony ridge containing these sockets.
GINGIVA (L. gum). = Connective tissue covered by mucosa.
ORAL EPITHELIUM PROJECTING DOWNWARD.

Fig. 2.

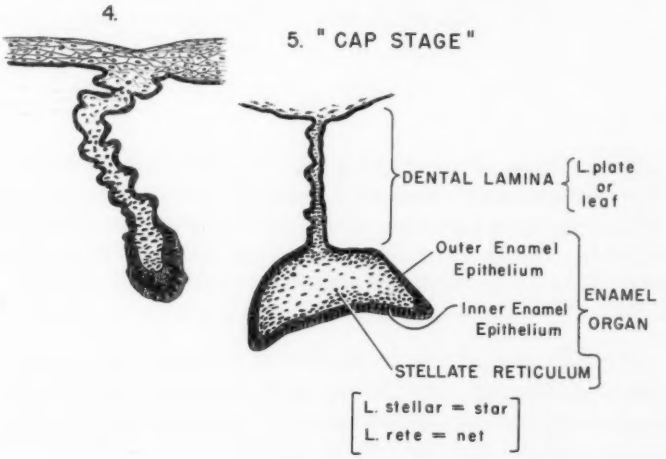
4.
5. " CAP STAGE"
DENTAL LAMINA
L. plate or leaf
Outer Enamel Epithelium
Inner Enamel Epithelium
ENAMEL ORGAN
STELLATE RETICULUM
L. stellar = star
L. rete = net

Fig. 3.

There now follows a condensation of mesenchymal tissue about this enamel organ (see Fig. 4). This occurs especially under the inner enamel epithelium, and gradually pushes upward, forming a nipple-like projection, called the dental papilla. As a result, the former "cap" now looks more like a bell, and this is, therefore, called the "bell" stage.

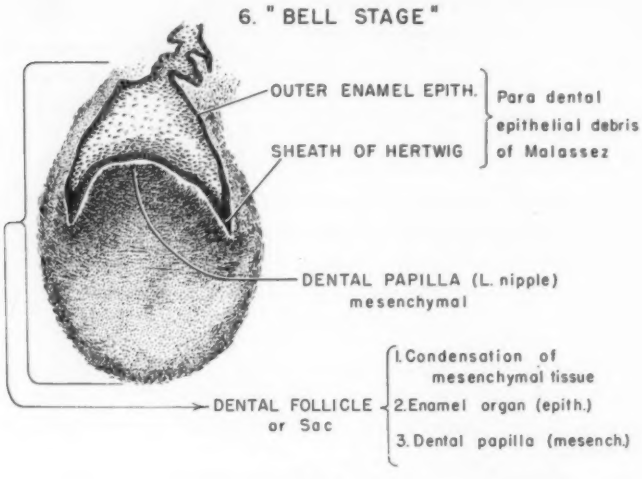

Fig. 4.

The juncture of outer and inner enamel epithelium forms a progressively more sharp edge. In three dimensions it can be visualized as the free edge of the "bell." This is the sheath of Hertwig. It and the outer enamel epithelium normally atrophy. They are collectively called the Paradental Epithelial Debris of Malassez.

With atrophy of the dental lamina and also with the formation of the epithelial debris, three structures are left isolated. These are the condensation of mesenchymal tissue, the enamel organ, and the dental papilla. They make up the primitive dental follicle or sac.

The inner enamel epithelium now develops the ability to form enamel (see Fig. 5). Its cells are, therefore, called ameloblasts (amelo, enamel) or adamantoblasts. Opposing them, a layer of mesenchymal cells develop the ability to lay down dentine. They are, therefore, called odontoblasts. The formation of odontoblasts and the deposition of dentine are dependent upon the presence of an enamel organ.

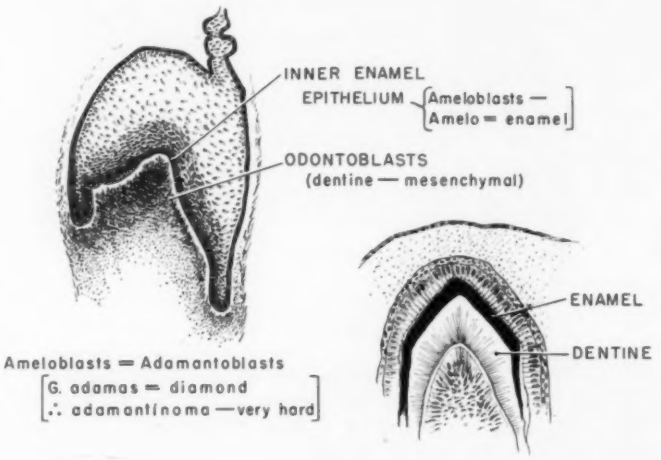

Fig. 5.

The ameloblasts and odontoblasts proceed to deposit enamel and dentine, the opposing layer of each becoming progressively thicker. It is noteworthy that the nuclei of the ameloblasts are *basal* with regard to the deposited enamel. In relation to the stellate reticulum, the nuclei are *non*basal, an important point in discussing adamantinomas later.

Mesenchymal connective tissue meanwhile invades the sheath of Hertwig, pushing it outward to become debris. With further condensation the connective tissue becomes the cementum (see Fig. 6), and there is now a crude representation of a tooth.

It can be visualized that with further differentiation, further projection of the tooth into the stellate reticulum and separation of the overlying oral tissues, the tooth erupts.

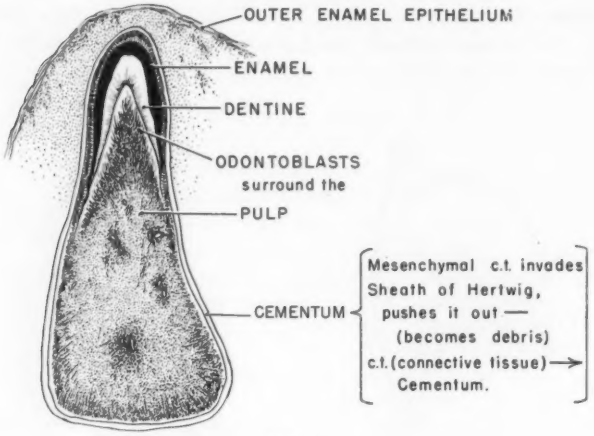

Fig. 6.

The process of development diagrammatically represented in Figs. 1 through 6 can be reviewed in photomicrographs made from serial sections of a fetal upper jaw. This fetus had a crown-rump length of 100 mm., representing about the fourteenth week of development.

The gradual thickening of oral epithelium is first observed (see Fig. 7) with the "bud" stage progressing to form a dental lamina. The latter progresses to the "cap" stage (see Fig. 8), and the next figure (see Fig. 9) shows a beginning "bell" stage.

These are tissues, therefore, whose histodifferentiation is not completed until after all permanent teeth have erupted. There is thus a long postnatal period in which growth disturbances may result in tumor and cyst formation.

Such growth disturbances may be due to inflammatory processes or nutritional deficiences, but especially neoplasia by the developing tissues or the epithelial debris.

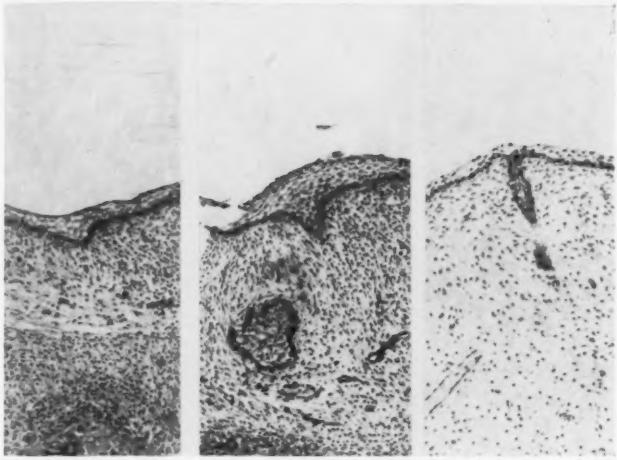

Fig. 7. Fetal jaw: Oral epithelium and "bud" stage.

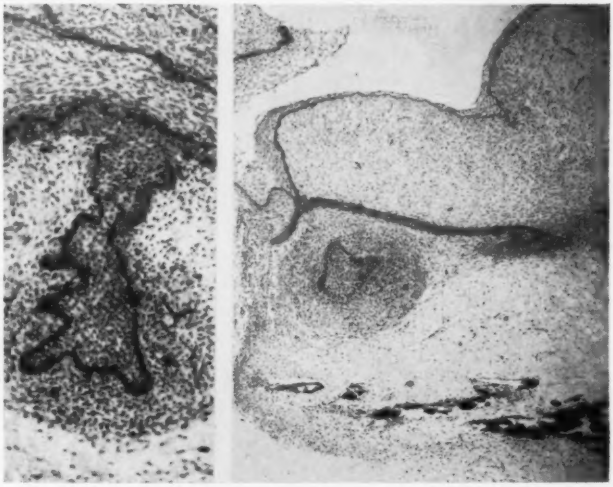

Fig. 8. Fetal jaw: Dental lamina and "cap" stage.

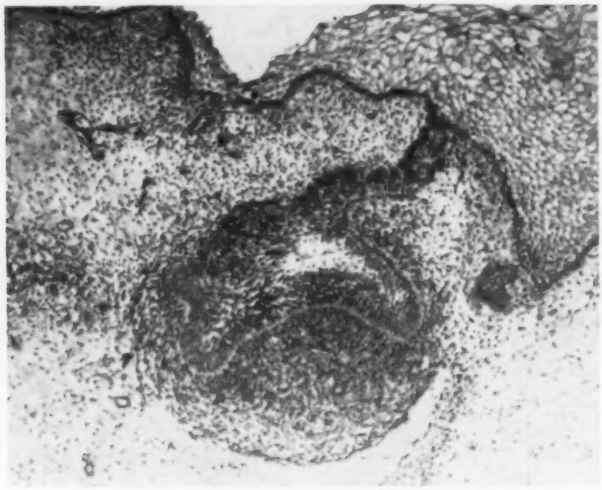

Fig. 9. Fetal jaw: Beginning "bell" stage.

POTENTIAL MALFORMERS.

Knowing the embryological and developmental components, the potential malformers can be tabulated (see Table 1). First, the epithelial debris includes that of the dental lamina, and the outer enamel epithelium and sheath of Hertwig of the enamel organ. Second, the dental follicle, the potential tooth, includes the epithelial enamel organ, and the mesothelial dental papilla and cementum. Components of the enamel organ are the epithelial debris of Malassez, the ameloblasts, and the stellate reticulum. Components of the dental papilla include the odontoblasts and the mesenchymal tissue proper.

CLASSIFICATION AND DISCUSSION OF ODONTOGENIC CYSTS AND TUMORS.

Knowing the potential malformers the odontogenic cysts and tumors can be classified (see Table 2) and after each list the structure of origin, *i.e.*, the structure whose maldevelopment or neoplasia resulted in the pathological entity.

Basically there are two types of odontogenic cysts: the follicular cyst and the radicular cyst. The follicular cyst, as its name implies, arises from the dental follicle or sac, and specifically from the sac-like enamel organ with its internal stellate reticulum. Many of the standard ear, nose and throat textbooks lack a clear definition of follicular and dentigerous cysts. As indicated in Table 2, the follicular cyst may be either simple or it may be dentigerous, *i.e.*, bearing a tooth. Knowing the embryology, the enamel organ can be visualized early in its development, dilating and becoming a simple, follicular cyst, or doing so at a later stage, after a tooth has partially or completely formed, and, therefore, forming a *dentigerous* follicular cyst.

TABLE 1.

POTENTIAL MALFORMERS

1. EPITHELIAL DEBRIS
   - a. Dental Lamina
   - b. Enamel Organ (Paradental debris of Malassez)
     - ___ Outer Enamel Epith.
     - ___ Sheath of Hertwig.

2. DENTAL FOLLICLE (potential tooth)
   - a. Enamel Organ (Epithelial)
     - ___ Epith. Debris
     - ___ Ameloblasts
     - ___ Stellate Reticulum
   - b. Dental Papilla (Mesothelial)
     - ___ Odontoblasts
     - ___ Mesenchymal tissue
   - c. Cementum (Mesothelial)

Follicular cysts are usually of the dentigerous variety, frequently associated with impacted lower third molars in young people. They may, however, occur in the maxilla. Modifying adjectives such as central or lateral refer to the position of the cyst in relation to the contained tooth crown. They may be multiple.

TABLE 2.

CLASSIFICATION OF ODONTOGENIC CYSTS AND TUMORS

A. CYSTS	ORIGIN
1. Follicular	Enamel Organ–stellate reticulum (epith.)
a. Simple	
b. Dentigerous (L. dens–tooth) (genere–to bear)	
central	
lateral	
multiple	
2. Radicular (root) (England–dental cyst)	Paradental debris of Mallassez (Sheath of Hertwig plus outer enamel epith.)

CLASSIFICATION OF ODONTOGENIC CYSTS AND TUMORS (cont'd.)

B. TUMORS	ORIGIN
1. Adamantinoma (ameloblastoma)	Enamel Organ (epith.) (–no enamel found)
2. Odontomas	
a. Enameloma	Inner Enamel epith.
b. Dentinoma	Dental papilla } mesoth.
c. Cementoma	C.T. } mesoth.
d. Mixed (ectod. and mesod.)	Varying parts of Dental Follicle

QUALIFYING TERMS:

Soft	Compound	(with teeth)
Calcified	Geminated	(L. geminatio – a doubling; twins)
Complex	Cystic	

The radicular cyst, as its name implies, occurs in the area of the root of a tooth, usually a diseased or devitalized tooth. The epithelial debris of Malassez, particularly the sheath of Hertwig, appears prone to inflammatory disturbance and cyst formation.

As indicated in Table 2, these cysts are of epithelial origin, embryologically from the parent stratified squamous oral epithelium. Their lining, therefore, is stratified squamous epithelium, occasionally flattened down by the pressure of the transudate or exudate filling the cyst into a simple, pavement epithelium.

The odontogenic tumors also include two main types: one is the adamantinoma or ameloblastoma; the other, the various odontomas or "tooth-tumors."

The adamantinoma arises from the enamel organ, an epithelial structure. It retains the undifferentiated character of this structure in that no enamel is found in the tumor tissue. This contrasts with the enameloma. As previously noted, the nuclei of the adamantoblasts, or ameloblasts, are toward the side of the stellate reticulum. This appearance of "nonbasal" nuclei is retained in the adamantinoma. It is sometimes described as resembling a gland turned inside out.

In one series of 379 cases of adamantinoma, 70 per cent appeared in patients in the age group of 10 to 35 years.[2] They were equally divided between the sexes. Adamantinomas occur much more commonly in the mandible than in the maxilla.

The local invasiveness of adamantinomas has been repeatedly emphasized, and deservedly so.

The odontomas include so-called enamelomas, dentinomas, cementomas, and tumors of mixed tooth constituents. The enamelomas are of epithelial origin, the dentinoma and cementomas, mesothelial; the mixed odontoma is the most common. Often the label used merely indicates the predominant constituent. Various qualifying terms are used to describe lesser characteristics.

TREATMENT.

Basically, the treatment of odontogenic tumors and cysts consists of surgical excision. This should include the offending tooth or teeth, in the case of dentigerous or radicular cysts. Simple marsupialization of the cyst, with or without a plastic button or similtar prosthesis to keep the orifice open, has been advocated, without the removal of the cyst lining. The claim is made that the cyst cavity will then granulate in. Such occurrence would be a unique phenomenon in the human body. Surgical diathermy to the bony surfaces of the defect helps insure complete removal of the lining membrane.

Of the odontogenic tumors, excision and cure is fairly readily obtained except in the case of adamantinomas. The invasiveness and locally-malignant nature of the latter cannot be overemphasized. In this condition surgical excision must be radical in nature, with a wide margin. The following case report illustrates this:

ADAMANTINOMA: REPORT OF A CASE.

B. P., a very lovable little four-year-old boy, was first seen in the Stanford Ear, Nose and Throat Clinic in March, 1951, complaining of displacement upward of the left eye, of four months' duration. Two weeks before coming to the clinic, X-ray examinations had revealed an abnormality of the left maxilla. Additional history is not pertinent.

On examination the only positive findings included a slight proptosis of the left eye, equal palpebral fissures, but a slightly higher position of the left cornea compared to the right.

Further X-ray studies (see Fig. 10) revealed a mass in the left antrum, absence of much of the lateral and inferior walls of the antrum as well as the floor of the left orbit. Tooth structures were noted in the mass.

On exploration via a Caldwell-Luc approach, the tumor seemed to have a capsule which could be separated from bone where present, and from residual periostum where the bone had been destroyed. Frozen sections determined the diagnosis of adamantinoma (see Fig. 11). (The resemblance of this tissue, with its stellate reticulum and darker epithelial elements, to the developing tooth structures shown in Figs. 8 and 9, is striking.) An inferior meatal window was fashioned and the incision closed.

Because of the notorious difficulty of complete removal of this type of tumor, the patient was then presented before the Stanford Tumor Board for advice as to the best therapeutic course. The Board advised multiple biopsies of the walls of the operative defect for evidence of persistent tumor. It was hoped that the left eye could be spared.

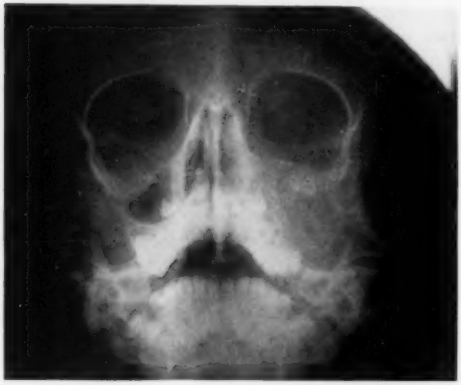

Fig. 10. Destruction of walls of left antrum by adamantinoma.

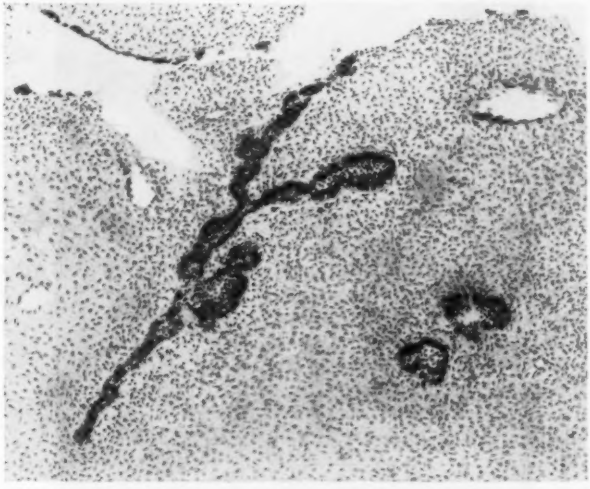

Fig. 11. Adamantinoma.

Fig. 12. Orbital contents and maxilla, removed en bloc.

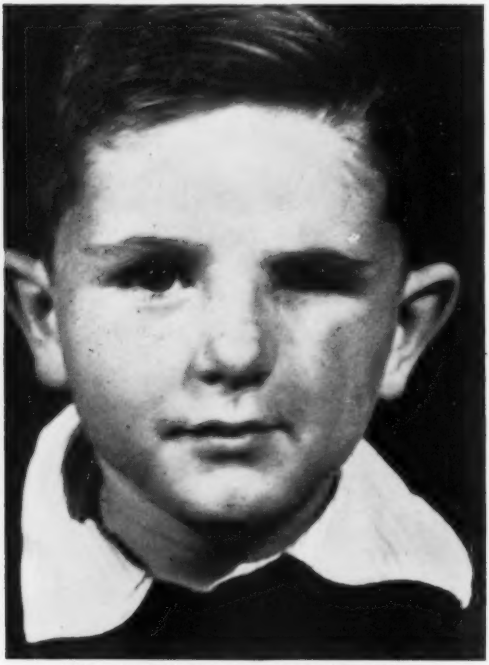

Fig. 13. Patient one year postoperative.

The surgical cavity was, therefore, re-entered and multiple biopsies taken. These showed extension of the adamantinoma to the lateral wall, floor, and medial wall of the antrum, and involvement of the left periorbita both anteriorly and posteriorly. The Tumor Board, therefore, recommended radical exenteration, including the left orbital contents, as necessary for cure.

The entire left maxilla, part of the zygomatic bone, the pterygoid process of the sphenoid and the entire orbital contents were then resected en bloc (see Fig. 12). The incision circled the palpebral fissure, extended downward along the side of the nose, then along the columella, separating the upper lip. After removal of the specimen the eyelids were sutured together, and the cheek flap and part of the surgical cavity were lined by a split thickness graft from the abdomen, held in place by sponge rubber packing.

Postoperatively, a dental prosthesis was fashioned to close the palatal defect. It has been modified several times with the growth of the patient. He is now one year postoperative (see Fig. 13) with as yet no sign of persistent or recurrent tumor.

SUMMARY AND CONCLUSIONS.

A knowledge of the terminology, nosology and embryology of tooth structures is essential for the understanding of odontogenic tumors and cysts. This basic information has been reviewed, and correlated with the formation, diagnosis and treatment of such tumors and cysts. The individual anlage have been tabulated. Therapy has been discussed, and the need for radical surgery for cure of adamantinomas has been stressed. A case report illustrates this type of therapy.

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TRACHEOTOMY IN BULBAR POLIOMYELITIS.

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Nearly all of the deaths in poliomyelitis occur in patients with bulbar involvement; therefore, any improvement in the management of this phase of the disease assumes great importance. During the past decade tracheotomy has been performed in hundreds of cases of bulbar poliomyelitis, and the literature now contains many favorable reports of its use.[1] There are other workers, however, who have had wide experience with this disease who contend that they have not seen cases requiring tracheotomy.[2,3] Even among those who advocate the operation there is a difference of opinion as to its indications and the time when the procedure should be carried out.[1]

Much of the confusion that exists is due to the fact that the pathology and pathological physiology of this disease is not too well understood. The problems involved and the literature have been reviewed quite completely by Galloway,[1b,1d,1i] Baker,[4] Cummings,[1j] Mitchell and Hill[1g] and Priest *et al.*[1c,1k]

In order to understand the clinical manifestations of the disease the following classification (Galloway and Seifert[1d]) is presented:

1. Spinal: Diaphragm and intercostal muscle paralysis.
2. Bulbar paralysis: Involvement of the swallowing mechanism and resultant accumulation of secretion, food and fluids, and vomitus in or over the airway.

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3. Bulbar-spinal: A combination of the above (1 and 2).

4. Other disturbances in rhythm and depth of respiration (so-called poliomyelitic encephalitis).

The use of tracheotomy in bulbar poliomyelitis is based upon the concept (Galloway[1b]) that most of the severe central effects result from anoxia and carbon dioxide accumulation due to secretional obstruction of the air passages. The direct action of the virus on the central nervous system is considered less important than the effect of anoxia. Tracheotomy is resorted to in order to clear the airway and relieve obstruction.

Brown[5] states that "we agree in Minneapolis as to the extreme importance of anoxia and maintenance of an adequate airway in poliomyelitis; however, we disagree in part with Dr. Galloway. We have seen cases showing definite and severe inflammatory involvement of the respiratory center and the circulatory center in the medulla. These cases have been studied histologically and have been reported in the literature. From 1946 to 1948, we saw 318 cases of bulbar poliomyelitis with a mortality of 34.2 per cent, in spite of every effort made on our part to maintain an open airway."

The following are Brown's mortality statistics:

Pure cranial nerve involvement—mortality 5.6 per cent.

Respiratory center involvement—mortality 70 per cent.

Circulatory center involvement—mortality 85 per cent.

Relatively pure encephalitic symptoms—mortality 0.3 per cent.

Bulbar with involvement of diaphragm and intercostals—mortality 54 per cent.

It appears from the above discussion that bulbar poliomyelitis is not a simple disease entity. Many cases may be problems solely of respiratory obstruction, but others will show the more serious symptoms of medullary center involvement.

The indications for tracheotomy in poliomyelitis according to Galloway and Seifert[1d] are:

1. Progressive anoxia with secretion in the upper airway.
2. Unconsciousness or pronounced restlessness in a patient who does not respond to other treatment within a few minutes.
3. Pronounced restlessness or stupor in a patient in a respirator, even if the paralysis is apparently of a spinal type.
4. Fluid accumulation not otherwise certainly taken care of in a patient who requires a respirator.
5. Bilateral paralysis or spasm of vocal cords.
6. Rapidly progressive bulbar symptoms.
7. Grave signs of vasomotor failure.
8. Untrained or inefficient attendants, inadequate equipment or poor cooperation of the patient, with doubt that the airway will be kept constantly free of secretion.

Advantages of the use of tracheotomy in the care of these patients are: *1.* they can be placed in a respirator and will synchronize successfully (Galloway and Seifert[1d]), *2.* clearing of secretions from the lower airway is facilitated, and *3.* oxygen under positive pressure which has been humidified can be delivered through the tracheotomy tube. This delivery can be synchronized with the respirations and even with the respirator when necessary.

The following cases represent our experience with tracheotomy in poliomyelitis at the Kingston Avenue Hospital in the years 1949 and 1950. During those two years there were 1,237 patients admitted with poliomyelitis. Two hundred and three patients had bulbar or bulbar-spinal involvement and all of the deaths (89) occurred in this group. The mortality rate for the patients with bulbar involvement was 43.8 per cent and the mortality rate for all the cases (1,237) was 7.2 per cent.

Six tracheotomies were performed, and five of those operated upon died. The case reports of the six tracheotomized patients are presented on next page:

CASE REPORTS.

Case 1: A 16-year-old white male was admitted on Sept. 3, 1949, with a history of vomiting, headache and stiffness of the neck of 12 hours' duration. Examination revealed a slight nuchal rigidity and bilateral hamstring spasm. Spinal tap revealed 495 cells. He developed a rapidly ascending paralytic involvement, so that on Sept. 4 there was paralysis of both lower extremities and the intercostal muscles. A slight nasal twang was noted. On Sept. 5 he began to vomit continuously and became lethargic. Frequent suctioning was required. A Levine tube was passed and he was placed in an oxygen tent, and a tracheotomy was then performed. At the completion of the operation the color of the patient was described as "poor" and in spite of oxygen, suction and stimulants he never improved. He expired 15 minutes after the completion of the tracheotomy.

Autopsy Findings: "There is a tracheotomy wound in the midline of the neck extending to between the second and third cartilages. The tracheal mucosa about the wound is reddened. Within the tracheal and bronchial passages there is a moderate amount of gastric contents. The right lung weighs 290 gm., the left 250 gm. Both lungs are voluminous, hypercrepitant and emphysematous. They are pink in color and show a few patches of atelectasis, bilaterally, at the hilum." The diagnosis was emphysema and atelectasis of the lungs.

Case 2: A five-year-old white male was admitted on Sept. 12, 1949, with a two-day history of sore throat, fever and vomiting. On admission his temperature was 102° F. He had a rigid neck, bilateral hamstring spasm, occasional upper extremity tremulousness and a suggestion of a nasal twang. Spinal fluid showed 87 cells. On the day after admission he developed a large amount of mucus in the throat, respiratory distress and tremor. There was reddening of the pharynx with pooling of mucus. The uvula deviated to the left and the right palatine arch was widened. Direct laryngoscopy revealed abductor weakness of the left vocal cord. A tracheotomy was performed. At the completion of the operation the patient's condition was good. His color was good and there was no dyspnea. Within five hours, however, there was rapid deterioration of his condition with the development of quadriplegia, intercostal and diaphragmatic paralysis. He was placed in a respirator and oxygen tent. There was no difficulty in keeping the tracheotomy tube free from secretions. Despite this therapy he went into a coma, had a convulsion and expired about eight hours after the tracheotomy.

Autopsy Findings: Right lung weighs 220 gm. and the left weighs 145 gm. Both lungs show hemorrhagic areas scattered throughout (left more than the right). There is a small amount of aspirated gastric contents in the lower trachea and bronchi.

Case 3: A 19-year-old white male was admitted on Sept. 17, 1949, with an onset three days previously of sore throat, headache and nausea. Vomiting, malaise and difficulty in swallowing commenced the next day. At the time of admission he was vomiting and his temperature was 102° F. There was a nasal twang and slight deviation of the tongue and uvula to the right. There was no stiffness of the neck. Spinal tap was done and the cell count revealed 95 cells. Later on the day of admission he developed difficulty in breathing and became very apprehensive. His face was markedly suffused and dusky. There was pooling of mucus in his throat. Tracheotomy was immediately performed. His color was good at the completion of the procedure. About two hours later, however, he

developed irregular respirations (like Cheyne-Stokes) and again became restless and apprehensive. His temperature was 102.6° F., blood pressure 130/90 and the pulse was of good quality but rapid. The tracheotomy tube was not obstructed and nothing was obtained on suctioning. His skin became cold and mottled. Despite oxygen and supportive treatment, he expired about 12 hours after the operation. No autopsy was obtained.

Case 4: An eight-year-old white male was admitted on Sept. 24, 1949, with a four-day history of nausea, vomiting and fever. For two days he had had back and neck spasm. He had a nasal twang for one day. Examination revealed a rigid neck, a nasal twang and deviation of the uvula to the right. On the following day his condition was worse. He showed weak intercostal function and the respirations were rapid and shallow but regular in rhythm. There was left deltoid weakness and a right facial paralysis. He developed difficulty in swallowing and clearing the hypopharynx of the rapidly accumulating secretions. He was oriented but lethargic. A direct laryngoscopy was performed and an accumulation of secretions in the epiglottic region was noted. There was a paralysis of the right vocal cord. An airway was introduced, and a tracheotomy was performed over it. The postoperative course was smooth. There was no further respiratory difficulty. Blocking of the tracheotomy tube was started one week after the operation, and he was decannulated on the eleventh postoperative day. On Oct. 24, 1949 (one month after admission) he was discharged to an orthopedic hospital for further treatment of a left deltoid weakness. Except for this, however, he was well and the pharyngeal and palatal muscles were functioning normally.

Case 5: A nine-year-old white male was admitted on Sept. 30, 1949, with a history that two days previously he had been beaten by three boys. The following day he was dizzy and nauseated. On the day of admission he was incoherent, he had vomited and had been unable to sleep. His temperature was 104.2° F. and he had nuchal rigidity, a right facial paralysis, palatine paralysis with inability to swallow, deviation of the tongue to the left, weakness of the right leg and absent abdominal reflexes. There was pooling of secretions in the pharynx, he was unable to speak and was periodically cyanotic. No spinal tap was performed. A direct laryngoscopy was done and excessive secretions were found in the hypopharynx. The glottic chink was half open with no abduction and sluggish adduction of the vocal cords. An airway was inserted and tracheotomy was performed. He was placed in an oxygen tent. Two hours postoperatively he seemed more responsive and his general condition was somewhat improved. Eight hours after the tracheotomy he became worse, he was cyanotic and the respirations were irregular and shallow. He did not respond to commands. His face was ashen, the lips and fingers cyanotic and a mottled erythema was seen over the trunk. His blood pressure was 120/80 and his pulse was 130. He expired 13 hours after the tracheotomy. No autopsy was obtained.

Case 6: An eight-year-old white male was admitted on Aug. 14, 1950, with a one-day history of headache, nausea and vomiting. A nasal twang was noted on the day of admission. He had a temperature of 101.2° F., good color and normal respirations. The palatal and pharyngeal muscles were immobile and there was regurgitation through the nose. Spinal tap showed 222 cells. The following day the patient had inspiratory stridor and difficulty keeping the pharynx clear of secretion. He appeared toxic and had anxious facies. Direct laryngoscopy revealed secretions in the hypopharynx. There was a good airway and no vocal cord paralysis. It

was felt that the secretions were causing a spasm of the glottis. An airway was inserted and a tracheotomy was performed over it. He withstood the procedure well and his breathing was made more comfortable. His pulse became stronger and he was able to doze off for a while. About five hours after the completion of the tracheotomy, however, he became cyanotic, his pulse became rapid and thready and his temperature began to rise. His airway was clear. Despite oxygen and supportive therapy his temperature went up to 108° F. and he expired about seven hours after the operation.

Comment: The patient in Case 1 was operated upon in the terminal stage of his illness when there was spinal, cranial nerve, circulatory center and encephalitic involvement. Autopsy revealed evidence of aspiration of gastric contents. Certainly if tracheotomy is expected to help, it should be done before the patient is moribund.

The other four patients who died were operated upon early while their general condition was good. In all of them there was pooling of secretions, requiring frequent suctioning to prevent obstruction of the airway and hypoxia. There was cranial nerve involvement, as indicated by palatal paralysis, and in some of the cases pharyngeal paralysis was recorded. Two of these patients had vocal cord paralysis, one unilaterally and the other bilaterally. In one patient the stridor appeared to be due to spasm of the vocal cords. No technical difficulty was encountered during the operations as they were all done in patients who were not in a respirator. Some of the trachcotomies were done over an airway. In none of these six patients did pneumothorax or mediastinal emphysema develop postoperatively. The immediate relief of dyspnea, cyanosis and restlessness after the operation proved that the obstruction had been overcome. No difficulty was encountered in keeping the tracheotomy tube clear of secretions and in none of these patients were there excessive secretions in the trachea and bronchi after the operation.

There was evidence of cardiovascular center involvement in four patients (Cases 1, 2, 3 and 6) and respiratory center involvement in one (Case 5). In Case 4 the disease was limited to spinal and cranial nerve involvement and we believe that tracheotomy saved this patient's life. Chart I is a summary of the findings in the six tracheotomized patients.

Brown and Baker[6] state that "the involvement of the respiratory regulating center generally develops somewhat later in the illness than do the cranial nerve palsies." It would seem to us, therefore, that in the patients upon whom we operated early we provided a good airway and oxygenation, but we did not alter the effect of the virus on the medullary centers, as suggested above, and this ultimately led to the death of the patient. On the other hand, had a tracheotomy not been performed, these patients would have probably died from the effect of anoxia and carbon dioxide accumulation before the full effect of the virus could be brought to play upon the medullary centers.

Only one of these patients was placed in a respirator post-operatively. Although oxygen was given to these patients after the operation, none was administered by positive pres-

CHART I.

SUMMARY OF TRACHEOTOMIZED CASES.

	Case 1.	Case 2.	Case 3.	Case 4.	Case 5.	Case 6.
Lived or Died	Died	Died	Died	Lived	Died	Died
Day of Disease Before Operation	3rd	3rd	4th	5th	4th	3rd
Bulbar (B) or Bulbar-spinal (BS)	BS	BS	B	BS	B	B
Palatal Paralysis	Yes	Yes	Yes	Yes	Yes	Yes
Pharyngeal Paralysis	?	?	?	Yes	Yes	Yes
Vocal Cord (abductor) Paralysis U-unilateral; B-bilateral	*	U	*	U	B	None
Spasm of Vocal Cords	*	No	*	No	No	Yes
Pooling of Secretions	4+	4+	3+	4+	3+	4+
Condition at Time of Tracheotomy	Poor	Good	Good	Good	Fair	Good
Condition Immediately After Tracheotomy	Expired	Good	Good	Good	Fair	Good
Time of Onset of Deterioration Post-operatively (hours)	Immediately	5	2	----	8	5
Expired Post-operatively (hours)	½	8	12	----	13	7
Type of Death CV- Cardiovascular failure R - Respiratory failure	CV	CV	CV	----	R	CV

*--Not Examined

sure through the tracheotomy tube. On the basis of our present experience we can only speculate as to whether our results would have been better if the respirator and humidified oxygent under positive pressure had been used in the patients who died.

The survival rate of 16.6 per cent (one out of six) for the patients with bulbar poliomyelitis undergoing tracheotomy was lower than that for all the patients in this series with bulbar involvement, which was 57.2 per cent (114 out of 203). These figures merely indicate that the more desperately ill patients are operated upon, and a fair percentage of those who survive owe their lives to the operation. It would be entirely wrong to attribute the increased mortality rate to the tracheotomy itself. Chart II indicates that the experience of other workers coincides with ours.

CHART II.

MORTALITY RATE.

	Bulbar Cases	Deaths	Number of Tracheotomies	Deaths
Miller and Buck[1h]	388	101	198	77
Mitchell and Hill[1g]	39	4	10	4
Strobel and Canfield[1f]	17	6	10	5
Priest *et al.*[1k]	169	65	56	31
Friedman and Gilbert	203	89	6	5

SUMMARY.

1. Tracheotomy may be a life-saving procedure in bulbar poliomyelitis.

2. On the basis of our limited experience, we believe that the operation is indicated in the presence of:

a. Bilateral vocal cord paralysis.

b. Secretion obstructing the airway, especially when associated with unilateral vocal cord paralysis or spasm of the larynx.

3. It is preferable to perform the operation before there is evidence of medullary center involvement.

4. In spite of an early and well-functioning tracheotomy, involvement of the medullary centers, with respiratory and circulatory failure, does follow.

5. This report covers our experience with six patients with bulbar poliomyelitis who underwent tracheotomy, five of whom died. We believe that the operation saved the life of the patient who survived.

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17 Pine Street.

1404 President Street.

LARYNGOSCOPE MODIFIED FOR OXYGEN INSUFFLATION.†

MERRILL W. MICHELS, M.D.,
Oakland, Calif.

The modification of the standard Jackson and Holinger laryngoscope with a channeled tube* so that oxygen can be insufflated during laryngoscopic procedures has provided a much more useful instrument.

The coincidental use of oxygen with sodium pentothal and curare in performing laryngeal operations prevents hypoxia and allows for greater latitude of procedure. Good preliminary anesthesia is essential; morphine and either atropine or scopalamine should be administered prior to pentothal to check the manifestations of parasympathetic stimulation such as laryngeal spasm or cough. Sodium pentothal produces unconsciousness but does not inhibit reflex muscular activity; this reflex is depressed by preliminary medication. The reflex activity of the larynx can be satisfactorily blocked by the use of a topical anesthetic as pontocaine 2 per cent. Since the margin between production of relaxation and respiratory paralysis is narrow with curare, artificial respiration with oxygen is often necessary.

The direct channeling of oxygen to the glottis while doing laryngoscopic procedures has obviated many of the disagreeable features of pentothal and curare.

It has been my practice to introduce routinely an endotracheal tube on completion of the laryngoscopic work and to

*These instruments were specially made by George P. Pilling and Son Co., Philadelphia, Pa.

†From the Department of Otolaryngology, Permanente Hospitals.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication, March 20, 1952.

leave it *in situ* until the patient has well reacted. The help of a competent anesthetist is a requisite.

AMERICAN MEDICAL WRITER'S ASSOCIATION.

Dr. Walter C. Alvarez, of Chicago, nationally known medical editor, author and teacher, has been honored as recipient of the first Honor Award to be given by the American Medical Writer's Association. The award, consisting of a gold medal and certificate, was presented to Dr. Alvarez at the dinner on the occasion of the ninth annual meeting of the association at the Jefferson Hotel, St. Louis, Oct. 1. Dr. Alvarez is Editor-in-Chief of *Modern Medicine*, Professional Lecturer at the University of Illinois College of Medicine.

The Distinguished Service Award of the association was given to Dr. Harold Swanberg, of Quincy, Ill.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

Dr. Philip S. Hench, of Rochester, Minn., internationally known internist and research worker, has been honored by the Mississippi Valley Medical Society as its Honor Award recipient for 1952. The award, consisting of a gold medal and a certificate, was presented Dr. Hench at the banquet on the occasion of the seventeenth annual meeting of the society at the Jefferson Hotel, St. Louis, Oct. 2. Dr. Hench is Professor of Medicine, Mayo Foundation, University of Minnesota, and a 1950 Nobel Laureate in Physiology and Medicine.

Dr. Willard O. Thompson, of Chicago, Ill., was given the Distinguished Service Award of the society.

In Memoriam

MOSES DAVID LEDERMAN, M.D.,

1868-1952.

With the passing of Dr. M. D. Lederman, of New York City, on Sept. 7, 1952, the medical profession and the community lost a pioneer physician, a gifted author and editor and a great personality.

Dr. Lederman received his M.D. degree from the medical department of the University of Pennsylvania in 1889, and his postgraduate study was continued in the clinics of London, Rome, Vienna, Berlin and Egypt. Shortly after completing his studies he became associated with Seiler (Seiler's Cartilage and Seiler's Tablets) and within two years he was entrusted with the major responsibilities both at Dr. Seiler's office and at several of the most important otolaryngological clinics in New York City.

His professional development was rapid. Very early in his career he revealed unusual surgical skill and by the turn of this century he was recognized as an outstanding leader in his specialty. About this time he was appointed Associate Professor of Otolaryngology at the Poly-Clinic Hospital and at the same time served as Chief of Clinics at Manhattan Eye and Ear Infirmary and at Mount Sinai Hospital.

In 1895, he became a charter member of the American Laryngological, Rhinological and Otological Society and he was the first otolaryngologist to be invited to the editorial staff of THE LARYNGOSCOPE when it was established in 1896, in which capacity he served for 56 years. He was an ardent supporter of this journal throughout his professional life, and his long association with it was a source of great pride to him and of great service to the journal.

In 1908, Dr. Lederman was chosen Senior Otolaryngologist to Lebanon Hospital and served on its medical board as member, officer and finally as consultant until his death. He derived great satisfaction from his association with this hospital for these many years, and he counted every member of the medical staff and Board of Managers as his friends.

In an era antedating specific drugs and antibiotics he developed techniques and methods in the management of suppurative ear conditions and described these methods in numerous publications. He was in constant correspondence with specialists all over the country.

Dr. Lederman belonged to a generation of leaders, trail blazers and teachers who made worthwhile contributions to medical history and we are indebted to him for his share in these contributions.

It has been said that every practicing physician renders psychiatric services by his manner and personality, and Dr. Lederman's personality was most engaging and genial.

Tall, distinguished looking, with resonant voice and a ready smile, he was popular with all he met; his love for his fellow men and his cheerful disposition were very obvious, and people were readily attracted to him. He had a word of cheer for everyone, and on all occasions his warm-heartedness was evident in his speech and gesture. He never grew old in spirit, and he was especially popular with the young.

On his visits to the hospital in recent years he was the center of attraction. He was an excellent raconteur and you could see him surrounded by a group of eager listeners, both young and old, recounting most vividly some incident which, he said, occurred in his long and active career. These events were inexhaustible and were rarely repeated in the same way except for the twinkle in his eye and the dimple in his left cheek. These stories were frequently punctuated with a chorus of laughter. His subtle sense of humor was used effectively to dissipate tension or to create an atmosphere of hopefulness with his patients.

At the operating table he was a skillful technician and an excellent teacher. To the bedside he brought comfort and cheer; to the younger physicians he gave encouragement and inspiration, and to his colleagues, warmth, friendship and understanding.

We shall remember him as a warm-hearted friend, and as a kind, considerate fellow-worker. His absence will be keenly felt.

D. G.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

October 1, 1952.

Audicon Models 400 and 415.

Manufacturer: National Earphone Co., Inc., 20-22 Shipman St., Newark 2, N. J.

Auditone Model 11.

Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67.

Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chcago, Ill.

Beltone Symphonette; Beltone Mono-Pac Model M.

Manufacturer: Beltone Hearing Aid Co., 1450 W. 19th St., Chicago, Ill.

Cleartone Model 500; Model 700; Cleartone Regency Model.

Manufacturer: American Sound Products, Inc., 2454 S. Michigan Ave., Chicago 16, Ill.

Dahlberg Model D-1; Dahlberg Junior Model D-2.

Manufacturer: The Dahlberg Co., 2730 W. Lake St., Chicago 16, Ill.

Dysonic Model 1.

Manufacturer: Dynamic Hearing Aids, 43 Exchange Pl., New York 5, N. Y.

Electroear Model C.

Manufacturer: American Earphone Co., Inc., 10 East 43rd St., New York 17, N. Y.

Gem Hearing Aid Model V-35; Gem Model V-60.

Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.

Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico UE-Atomeer; Maico Quiet Ear Models G and H; Maico Model J.

Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis 1, Minn.

Mears (Crystal and Magnetic) Aurophone Model 200; 1947—Mears Aurophone Model 98.

Manufacturer: Mears Radio Hearing Device Corp., 1 W. 34th St., New York, N. Y.

Micronic Model 303; Micronic Model "Mercury"; Micronic Star Model.

Manufacturer: Micronic Co., 727 Atlantic Ave., Boston 11, Mass.

Microtone T5 Audiomatic; Microtone Classic Model T9; Microtone Model T10; Microtone Model T612; Microtone Model 45.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Cub Model C; National Cub Model D (Duplex); National Standard Model T; National Star Model S; National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 815 S. Hill St., Los Angeles 14, Calif.

Otarion Model E-4; Otarion Models F-1, F-2 and F-3; Otarion Model G-2; Otarion Model G-3.

Manufacturer: Otarion Hearing Aids, 159 N. Dearborn St., Chicago, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny-Mite); Paravox Model XTS (Xtra-Thin); Paravox Model Y (YM, YC and YC-7) (Veri-Small).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Permo-Magnetic Multipower; Radioear Permo-Magnetic Uniphone; Radio Ear All Magnetic Model 55; Radioear Model 62 Starlet; Model 72.

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Rochester Model R-1; Rochester Model R-2.
Manufacturer: Rochester Acoustical Laboratories, Inc., 117 Fourth St., S.W., Rochester, Minn.

Silvertone Model J-92; Silvertone Model P-15.
Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.
Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.
Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Model 700; Sonotone Model 900; Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

Superfonic Hearing Aid.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Televox Model E.
Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 97; Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 1700.
Manufacturer: Telex, Inc., Telex Park, Minneapolis 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster Model Royal; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill.

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Western Electric Models 65 and 66.
Manufacturer: Audivox, Inc., successor to Western Electric Hearing Aid Division, 259 W. 14th St., New York 11, N. Y.

Zenith Model 75; Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal.
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices employ vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).
Manufacturer: A. M. Brooks Co., 64 S. Bonnie Brae St., Los Angeles 5, Calif.

Aurex (Semi-Portable).
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.
Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.
Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

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President: Dr. Albert C. Furstenberg, University Hospital, Ann Arbor, Mich.
Vice-President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Me.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Ill.
Meeting: Roosevelt Hotel, New Orleans, La., May 1-2, 1953.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2, Pa.
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AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

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Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
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MID-WINTER.

Triological Council Meeting—Hotel Syracuse, Syracuse, N. Y., 9 a.m., Jan. 6, 1953.

Eastern Section Meeting—Hotel Syracuse, Syracuse, N. Y., Jan. 7, 1953.

Southern Section Meeting—Andrew Jackson Hotel, Nashville, Tenn., Jan. 12, 1953.

Middle Section Meeting—The Drake, Chicago, Ill., Jan. 19, 1953.

Western Section Meeting—The Elks Club, Los Angeles, Calif., Jan. 24, 1953.

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AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Palmer House, Chicago, Ill., Oct. 6-10, 1952.
Meeting: Roosevelt Hotel, New Orleans, La., Apr. 21-25, 1953.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

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Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1, Md.
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Secretary: Dr. Frasier Williams.
Treasurer: Dr. John Louzan.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

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Meeting:

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Justo M. Alonzo, Montevideo.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fourth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. Ricardo Tapia Acuna, Mexico City.
Time and Place: January, 1954, Mexico City.

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THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

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For Otolaryngology, Charlottesville, Va., Nov. 21-22, 1952.
Spring Meeting: Hot Springs, Va., May, 1953.

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Time: 6:00 P.M., fourth Monday of each month from September to May, inclusive.

THIRD LATIN AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

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UNIVERSITY OF PENNSYLVANIA
Graduate School of Medicine
36th and Pine Streets
Philadelphia, Pennsylvania

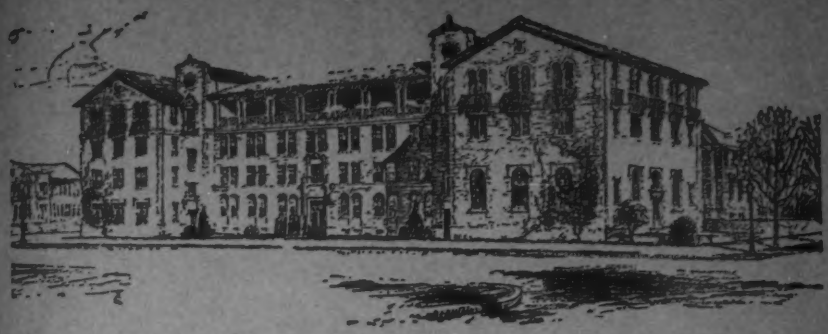
WASHINGTON UNIVERSITY SCHOOL OF MEDICINE
Euclid Avenue and Kingshighway
St. Louis 10, Missouri

TULANE MEDICAL SCHOOL
1430 Tulane Avenue
New Orleans 12, Louisiana
at Tulane Medical School and
Eye, Ear, Nose and Throat Hospital

NEW YORK UNIVERSITY
Bellevue Medical Center
Post-Graduate Medical School
477 First Avenue
New York 16, New York

Basic Sciences in Otolaryngology
September through June

*Our subscribers are asked to send us information on other institutions giving such courses.



Central Institute for the Deaf

NATIONAL RESIDENTIAL AND DAY SCHOOL FOR THE DEAF AND DEFECTIVES IN SPEECH

Approved by Advisory Council of Foremost Ear Specialists and Educators

New fire-proof buildings beautifully located opposite Forest Park. Modern Dormitories and Equipment. Best home environments. Pupils constantly in care of teachers or experienced supervisors.

ORAL SCHOOL FOR DEAF CHILDREN

C. I. D. offers all advantages of exclusively Speech Training and expert medical supervision for both Resident and Day Pupils.
Nursery School (2 years of age) through the Elementary Grades.

ACOUSTIC TRAINING FOR CHILDREN WITH RESIDUAL HEARING

Salvaging of Residual Hearing is a specialty of C. I. D. The Acoustic Method was created here. Group and individual hearing aids used for class instruction at all grade levels.

LIP-READING INSTRUCTION

Private and Class Instruction for Hard-of-Hearing Adults and Children.
Conversational Classes for advanced pupils. Speech conservation stressed.

CORRECTION OF SPEECH DEFECTS

Private and Class Instruction for children with normal hearing and delayed speech or defective speech.
Resident and Day Pupils (2 years of age through Elementary Grades).
Private Instruction for Adults.
Correction of Imperfect Phonation, Imperfect Articulation, Aphasia, Stuttering.

TEACHERS TRAINING COLLEGE

Two years of Training following a professional curriculum for applicants with adequate college qualifications. Graduates qualify for degrees of Bachelor of Science in Education or Master of Science in Education from Washington University. Graduates prepared to teach both the deaf and speech defective.

DR. MAX A. GOLDSTEIN, Founder DR. HELEN SCHICK LANE, Principal

818 S. KINGSHIGHWAY 10, ST. LOUIS, MO.

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